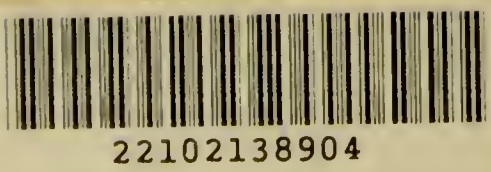
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DISEASES OF THE DIGESTIVE SYSTEM

MODERN CLINICAL MEDICINE

DISEASES

OF THE

DIGESTIVE SYSTEM

EDITED BY

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AN AUTHORIZED TRANSLATION FROM "DIE DEUTSCHE KLINIK" UNDER THE GENERAL EDITORIAL SUPERVISION OF

JULIUS L. SALINGER, M.D.

WITH FORTY-FIVE ILLUSTRATIONS IN THE TEXT

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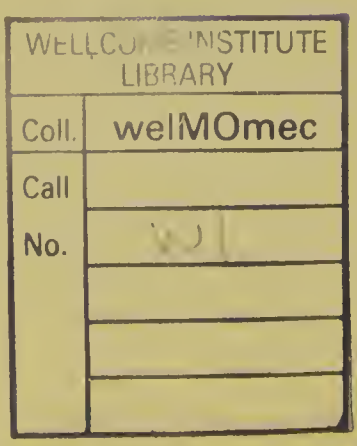

INTRODUCTION

The advanees of chemistry in the last few decades have placed the diagnosis and treatment of Digestive Diseases upon a firm foundation. Much that was formerly purely theoretical and based upon hypothesis has now beeome almost absolute knowledge. The study of metabolism has lifted the veil of mystery from mueh that was obscure. In the investigation of diseases of digestion, in which physiology, chemistry, and pathology are so intimately connected, the practitioner has received valuable aid in relieving suffering humanity. In practice, perhaps the greatest benefits may be eonveyed by the correetion of even the most insignificant alimentary errors.

In no organ of the abdominal cavity, and perhaps of the entire body, are the functions so manifold and diverse as in the pancreas. Text-books dating back a few decades scarcely mentioned the name of the organ, not to speak of a reference to its diseases. Thanks to the labors of investigators quite a literature is to-day extant, and the search-light of diagnosis is beginning to be thrown upon maladies of this important digestive organ.

This volume treats not only of these subjects, but a new study has been opened, or, more properly, revived; namely, the investigation of the feces. What the examination of the sputum is to disease of the lungs, what the examination of the urine is to disease of the kidney and to disorders of metabolism, what the analysis of the gastric contents is to the diagnosis of disease of the stomaeh, the macroscopic, mieroscopie, bacteriologic and chemical investigation of the feces is to disease of the intestines.

Abdominal surgery has aided much in the elucidation of many of these problems, and although we have reached but the first step of the ladder in the ascent of the tree of knowledge, much has been learned and still more may be expeeted.

EDITOR'S PREFACE

To-day diseases of the Digestive Tract stand in the forefront of subjects which interest the practitioner and the surgeon.

Many of the diseases included in this volume lie on the borderland of medicine and surgery.

This volume includes articles from many of the most eminent men of Europe, specialists in internal medicine and in diseases of the digestive tract.

The subjects are treated very fully and at the same time in a concise and practical manner. The modern methods of examination, including physical and chemical measures, are clearly set forth, which will enable the practitioner to apply them with the same ease that he may make a physical examination of the chest and a chemical and microscopic uranalysis.

The diagnosis of the various diseases is fully discussed and the treatment, including the dietary, is satisfactorily full and complete. Indeed, the subjects are so fully treated that the editor found it inexpedient to add to or to modify the text to any important extent.

The translator has done the work well, with the result that the text is smooth and interestingly readable.

Frank Billings.

100 State Street, Chicago.

LIST OF CONTRIBUTIONS

Stenosis of the Esophagus. By TH. ROSENHEIM, Berlin.
The History and Clinical Indications of Gastric Lavage. By W. FLEINER, Heidelberg.
Functional Diseases of the Stomach. By H. LEO, Bonn.
The Diagnostic and Therapeutic Significance of Secretory Disturbances of the Stomach. By H. STRAUSS, Berlin.
Diagnosis and Treatment of Gastric Dilatation. By F. RIEGEL, Giessen.
Gastric Ulcer and Gastric Hemorrhage. By C. A. EWALD, Berlin.
Gastric and Intestinal Carcinomata. By J. BOAS, Berlin.
Displacements of the Abdominal Viscera and of the Heart. By F. HIRSCHFELD, Berlin.
Symptomatology of the Diseases of the Pancreas. By L. OSER, Vienna.
Jaundice and Hepatic Insufficiency. By O. MINKOWSKI, Cologne.
Chronic Inflammation of the Liver. By E. STADELMANN, Berlin.
Neoplasms of the Liver and Biliary Passages. By FR. KRAUS, Graz.
Echinococcus of the Liver. By E. STADELMANN, Berlin.
Gall-stones. By E. NEUSSER, Vienna.
Acute Diffuse Peritonitis, Appendicitis, and Perityphlitis:
I. Diffuse and Circumscribed Peritonitis. By O. VIERORDT, Heidelberg.
II. Chronic Perityphlitis. By J. BOAS, Berlin.
Examination of the Feces. By J. STRASBURGER, Bonn.
Diarrhea, Intestinal Catarrh, and Intestinal Tuberculosis. By W. FLEINER, Heidelberg.
Constipation and Hemorrhoids. By J. BOAS, Berlin.
Mucous Colic and Membranous Intestinal Catarrh. By G. HOPPE-SEYLER, Kiel.
Intestinal Constriction and Intestinal Occlusion. By H. NOTHNAGEL, Vienna.

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DISEASES OF THE ESOPHAGUS AND OF THE STOMACH

STENOSIS OF THE ESOPHAGUS

By TH. ROSENHEIM, Berlin

ETIOLOGY

Diseases of the esophagus are, as a rule, infrequent. Almost invariably, however, they are of great importance, as an essential function of the organ is rapidly influenced, and because these processes occur in the immediate vicinity of the most vital organs, so that a dangerous transmission of the disease may readily take place. As the organ in question is one of the least sensitive ones, pain in disease of the esophagus is rare. In the main, there is but one symptom: *disturbance of deglutition.* This occurs in varying degrees of intensity in *all* esophageal affections, yet the severity of the causal process has no decisive influence upon the degree; for the hindrance in deglutition may be slight in carcinoma, and, on the contrary, it may be high-graded in small erosions, or in derangement of innervation. The basis for most disturbances of deglutition is a *stenosis of the lumen,* which may arise from manifold causes, and to which this article will be exclusively devoted. The majority of diseases occurring in the esophagus must be more or less considered; this will broaden our theme to a great extent, but we shall confine ourselves well within the required limits if we emphasize only the most important features from a *diagnostic* and *therapeutic standpoint.* The researches of the last few years, and especially since the introduction of esophagoscopy, have revealed much that is new and important.

The most frequent and, practically, the most significant cause of stenosis of the esophagus is *cancer;* other diseases are rarely mentioned, at most, here and there, stricture due to swallowing of caustics. But the forms of stenosis are by no means exhausted with this, and the spastic stenoses, particularly, call for more thorough consideration than was formerly devoted to them. *Disturbances in innervation play an important part in the origin of derangement of deglutition.* Yet it is often extraordinarily difficult accurately to measure the part which they play in the causation of the symptoms of the disease, as they rarely occur alone, but usually are combined with other pathologico-anatomical changes in the esophagus or adjacent organs. We must, however, always endeavor to analyze as accu-

rately as possible the genesis of the difficulty in swallowing, as this is of decisive importance in the therapy.

In reviewing the morbid processes in the esophagus which come into question under the picture of stenosis, the enormous amount of material may be divided into two groups: In the first group we are dealing with abnormal processes *which have their seat in the organ itself,* i. e., they arise directly from the organ, and thus narrow its lumen; in the second group we consider the cases in which stenosis is due to *compression.* Here may be mentioned tumors of the mediastinum, of the vertebral column, goiter or enlarged lymph-glands, aortic aneurysm, peri-esophageal abscesses, and, finally, filled diverticula which produce stenosis by pressure.

To the first group belong: (a) neoplastic stenoses; (b) inflammatory and cicatricial stenoses; (c) spastic stenoses; (d) hereditary stenoses, and, lastly, (e) the obstruction to the propulsion of the ingesta which may be produced by the esophagus, in which sense foreign bodies that have been swallowed, polypi with long pedicles, and aphthous stomatitis may act.

SYMPTOMS

Let us now describe the *subjective and objective symptoms of stenosis of the esophagus in the various forms of the disease which are to be here considered,* and let us observe the differences in the manifestations presented by the several types. For this purpose it is best to analyze the **symptoms of cancer of the esophagus,** the affection that most frequently causes stenosis of the esophagus. How frequent carcinoma is can be definitely seen from statistics which show that about 60 per cent. of all diseases of the esophagus are of a malignant nature, according to other compilations even 90 per cent.; but from my own experience I think the latter figure somewhat too high. It may be remarked in passing that cancer of the esophagus occurs in different countries with variable frequency. For example, in Moscow unquestionably the cases are more numerous than in Berlin.

Difficulty or an impediment in deglutition, therefore dysphagia, is the most constant symptom of cancer of the esophagus. It is usually the first, also, which makes the patient anxious, and leads him to consult a physician. The disturbance in swallowing appears gradually as mild pressure, sometimes behind the corpus sterni and behind the larynx, sometimes in front of the stomach and noted upon swallowing solid food; later, solid masses, even if well masticated, pass only with the greatest difficulty or only simultaneously with fluid; often they are arrested; then comes a stage in which watery, pappy food can be forced into the stomach only with effort; it must be swallowed in small portions, slowly and carefully; finally, the passage is entirely occluded, transitorily or permanently, so that the patient faces starvation. Besides this gradual, insidious, progressive development of

dysphagia, which is the usual course, we observe in some cases in the midst of the best of health and without prodromes a sudden and decided impediment to deglutition. At other times, decided variation in the degree of passability of the esophagus may be noted. On some days the patient can apparently swallow anything, on other days there is an obstacle. Doubtless the condition of the nervous system here plays an important rôle, for I have repeatedly observed this in very nervous patients with carcinoma of the esophagus. With a prolonged course of the affection, amelioration may come by the purulent softening and ichorous decomposition of prominent cancer proliferations; the patient again becomes hopeful, but this improvement is only the precursor of the final catastrophe.

The following fact is also remarkable: The patients complain of increasing difficulty in deglutition, so that even fluids fail to pass, or only with great effort, but, in this condition it is always possible to pass esophageal bougies into the stomach. *This disproportion between the natural power of deglutition and the results of probing* is found in all the varied affections of the esophagus, but chiefly in *compression of the organ* and in *neuroses;* occasionally, however, it is also observed in flat carcinomata situated in the wall of the organ and not distributed in annular form, since these offer no marked resistance to the sound, which readily passes them, and they disturb the active deglutition only in a reflex manner. Thus *spasms* are caused especially at the upper portion of the diseased area, but they may also occur in normal portions of the canal. For example, as not infrequent complications, we may have spasms behind the larynx, provided the tumor is situated in the cardia, and, in addition to this, very disagreeable *sensations of constriction* are felt at the boundary line between the pharynx and esophagus, and these necessitate continued efforts at swallowing on the part of the patient; this forms a very usual complication in the various diseases of the esophagus, and particularly in carcinoma, immaterial what its situation. The reports of the patient as to the region in which he feels constriction and pressure are often inaccurate and misleading. Frequently he will state that the bolus is arrested in the neck, while the cancer is situated in the cardia, or vice versa; the distress is sometimes localized in the epigastrium, when the neoplasm has developed in the upper half of the esophagus. *Therefore, the origin of disturbances of deglutition is the combination of a series of factors.* At one time, *mechanical obstruction* such as is presented by the infiltration of the wall of the esophagus plays an important rôle; at another, we observe *increased sensitiveness of the nerves of the muscular apparatus* which, as soon as the food reaches the inflamed area, readily show abnormal reaction. This reaction is the result of irritation, and is usually manifest as a spasm at the seat of the affection or in distant areas. Added to this are disturbances due to *inflammatory processes* in the mucous membrane of the areas surrounding the cancer, alone or in combination with an *atonic-ectatic condition* of the organ

above the neoplasm. In regard to the last point, it must be remembered that *hypertrophy,* and also *dilatation,* may develop above the stenosed area, that the latter, however, only rarely is decided, as a prolonged and permanent stagnation of food does not readily take place. On the contrary, when the bolus is arrested, it is usually soon regurgitated. This regurgitation naturally occurs the more promptly the greater the disproportion between the narrowness of the stricture and the consistency of the food, as well as between the local sensitiveness and irritability of the food. As a rule, regurgitation occurs immediately after swallowing, or from a few minutes to a quarter of an hour after, rarely later. When delayed, however, dilatation is usually present. In this case, if the esophageal sound be used, masses of food may be brought up in which the absence of peptonization shows clearly that they have never reached the stomach. It is obvious that *processes of decomposition* must arise when the food remains for a long time in the esophagus; consequently the patients not rarely complain of a *fetid odor* from the mouth. But these processes may occur in an advanced stage of the disease even without a decided retention of food owing to the decomposition of the cancer, and thus give rise to offensive breath.

Besides food, and even independently of its introduction, a *foamy, mucoid fluid* having a fetid odor, is often ejected, and not rarely shows traces of blood. . This is the product of the inflammatory process which accompanies cancer of the esophagus, and which sometimes also extends to the pharynx and larynx. The combined effect of these irritative factors is *cough,* which often increases to extremely severe paroxysms which naturally distress the patient; added to this is the fact that bronchitis and tuberculous changes in the lungs commonly coexist with cancer.

Apart from the unpleasant sensation of pressure mechanically caused by the arrest of the *bolus, the affection may be painless for a long time or even permanently, for the esophagus is one of the least sensitive organs,* and in this malady only an implication of the mediastinum, the pleura, and the pericardium, the compression of important nerve plexuses in the vicinity, the implication of the vertebral column and the spinal cord, will be productive of pain. Then *boring, burning, lancinating pains* set in which accompany the act of deglutition or follow it, but may come on independently of the introduction of food, particularly at night. The pains are felt in the intercostal spaces, between the shoulder blades, in the epigastrium, in the throat and head; for example, frequently in the ear and in the extremities. It is obvious that by the metastatic distribution, and by the further growth of the cancer, manifold symptoms may be produced. I shall here mention, for example, *vasomotor and trophic disturbances in the nails;* also the occurrence of *inequality of the pupils;* almost always the left pupil is contracted. *Paralysis of the recurrent laryngeal nerve,* above all, deserves special mention. Unilateral paralysis of the vocal cord is a quite common symptom. As this may not cause any change in voice production,

it can be detected only by a laryngeal examination, and thus is frequently overlooked. Often, however, it is an early symptom, and may even be present when as yet the stenosis is scarcely obvious.

Dyspnea and attacks of pain resembling angina pectoris arise in the course of the disease either because the neoplasm posteriorly presses immediately upon the trachea, or upon both recurrent laryngeal nerves, or implicates the posterior plate of cricoid cartilage and its surroundings, in which case the nerve and the muscular substance of the dilators of the vocal cords are directly destroyed.

Perforation of the esophageal wall may take place without producing special symptoms, and this is true even if the formation of a *fistula between the esophagus and the trachea or the bronchi* has already occurred, provided that the opening is small and the esophageal stricture slight, so that food may pass the organ without being arrested; otherwise, severe *paroxysms of cough and marked dyspnea upon the ingestion of food* are the characteristic signs of this last-named complication. The paroxysms of cough cause the ejection of mucus stained with blood simultaneously with particles of food which have found their way into the respiratory apparatus; sooner or later, upon continued nutrition by the mouth, pneumonia or pulmonary gangrene occurs. If the fistulous passage is very narrow, and the stricture still relatively permeable, it is sometimes noted that thin fluids cause paroxysms of cough, while more compact food passes with ease.

Decided spontaneous *hemorrhages* from cancerous ulcers in the esophagus which cause hematemesis are rare symptoms of carcinoma. More frequent are fatal *internal* hemorrhages due to the *erosion of a vessel* with the advance of the process.

Concerning the difficulties on the part of the *gastrointestinal canal* and the general condition: At the onset of the affection the patients usually have a good *appetite,* and in some pitiable cases this continues until death. In the further course of the disease, as a rule, hunger becomes less pressing, while burning *thirst* almost invariably persists. Stubborn *constipation* is common. In advanced cases, after the food has reached the stomach, it sometimes produces a continuous *dull pain in the epigastrium,* not rarely accompanied by eructations and nausea. These dyspeptic symptoms have no direct significance in cancer of the esophagus. They are to be attributed to secondary inflammatory atrophic processes in the mucous membrane of the stomach which are sooner or later added, and the more rapidly, the closer the cancer lies to the stomach, and the sooner it implicates this organ.

It is obvious that *inanition and cachexia* will early develop in the clinical picture, according to the nature of the disturbances which the affection produces. At first the loss in strength is parallel with the duration of the disease and the degree of obstruction to deglutition. If it is possible to diminish, or even to remove, the latter, although only tem-

porarily, the patients, as a rule, rapidly recover and increase in weight. Exceptionally carcinoma of the esophagus, without any functional disturbance, causes extreme debility by a deleterious and toxic influence upon proteid metabolism.

Examination of the patient in the various stages of the affection furnishes results which are further modified according to the seat of the difficulty and the complications. *Inspection and palpation* frequently permit us to recognize *enlargement of the lymph-glands* in the supraclavicular and cervical regions; but the utilization of this finding necessitates caution, since quite marked enlargement of the glands is also found in scrofulotuberculous individuals, a moderate enlargement not infrequently in syphilis and catarrhal affections of the organs of the throat; it *may even be present without a recognizable disease.* If the cancer is situated in the upper portion of the esophagus it may be externally palpated as a *tumor* beside the trachea and the larynx, and if it attack the cricoid plate or the pharynx it may even be *seen* by the laryngoscope.

When the cancer is deeply located, and cannot be thus recognized, we must *investigate by means of the sound.* We should first determine that *no disease is present which contraindicates the use of the sound,* and then attempt to pass the stricture with a thick instrument (10 to 12 mm. in diameter). After it is within the esophagus it should be *slowly and carefully* forced; if resistance is met with, it must be overcome by gentle means, but boring should be avoided; upon withdrawal *the length of the narrow portion from the teeth should be carefully noted.* It will frequently happen that the figures gained in this manner will vary several centimeters upon different days—this is explained by the fact that the sound enters to a different depth, or the point is embedded in the stricture—nevertheless, in spite of these variations, they enable us to locate the constriction, but give us no idea of its extent. The resistance that the sound meets may be in the upper, in the middle, or in the lower part of the malignant neoplasm. It is a valuable diagnostic aid *carefully to examine the fenester of the sound.* In this we frequently find remains of food, blood, pus, mucus, and sometimes even *shreds of tissue* which under the microscope permit the exact recognition of the disease. If it be impossible to recognize the characteristic cancer cell nests in the investigation of the fresh preparation, then the suspicious portion may be examined by the stroma preparation of sections after hardening in alcohol, or, a more rapid process, the frozen section with the microtome and its staining (for example, with picro-carmin).

After the presence of a stenosis has been determined by means of a thick sound, we should attempt to *pass through the narrow passage with more slender instruments;* frequently it will be impossible to reach the stomach with the finest instruments, although pappy and, occasionally, even solid food still pass the stricture. The explanation of this is either that

the point of the sound is caught in a diverticulum above the narrowed area, or the canal of the stricture is not straight but follows an irregular, spiral course. On the other hand, the patients may complain of difficulty in swallowing food in a certain area of the esophagus, yet the largest sound finds no resistance in its entire course to the stomach; *in cases of beginning cancer formation, particularly, the results of examination may be negative.* Here the latter course must explain the case, or, if an *early diagnosis* is desired, we must investigate by means of *the esophagoscope.* The narrowed area which has been revealed by the sound, even in advanced cases, may be entirely unrecognizable if extensive ulceration and rapid destruction have occurred; but here also the esophagoscope gives the desired information.

The esophagoscopic pictures show great variations, which certainly depend upon *the stage* of development of the neoplasm, and, above all, *the nature of the growth.* The picture which presents itself is entirely different when *cauliflower-like proliferations* enter into the lumen, or, on the contrary, when only *an induration* of the wall has occurred, when *ulcer formation* has taken place, when *the mucous membrane is still retained,* when *the narrowing* is very decided, or when it is very slight.

The cancerous protuberances which narrow the space, if accurately investigated, cannot be confounded with anything else; there is no process in the esophagus except carcinoma that runs its course with the formation of these *prominent, whitish-grey to greyish or dirty greyish-yellow nodules permeated by punctiform hemorrhages.* The finding of a prominence covered with mucous membrane, however, may be of varying significance.

Even in advanced cases of carcinoma the stenosis is generally not due to cancerous masses which have proliferated into the lumen, but is due to *infiltration,* and this, viewed from above, presents itself as a swelling or a round tumor about the size of a cherry with *well-retained mucous membrane,* while ulceration or splitting of the surface cannot be detected because it occurs only toward the inner wall, therefore, arises in the lumen of the stricture. Frequently, above these protuberances the mucous membrane appears paler than normal, sometimes more yellow or, cyanotically, bluish-red, occasionally even loosened and markedly reddened. Worthy of note are the *epithelial thickenings* in different forms (streaks, flakes, etc.), which resemble leukoplakia of the tongue, and are found in the immediate surroundings of the morbid focus. At other times *whitish, papillary excrescences,* resembling pointed condylomata, or papillæ consisting of several whitish papillary proliferations, are met with in front of the narrowed area, frequently situated upon a perfectly normal mucous membrane. These last mentioned changes are *not pathognomonic of carcinoma;* we also meet with them in other conditions.

Not rarely the wall is deeply infiltrated only in the cancerous mass; then the formation of the above-mentioned one-sided protuberance does

not occur, but frequently there is an *annular* narrowing, with increasing constriction downward. We can then pass the tube as far as this rigid area, but no farther, and we descry from above a funnel-shaped passage with mucous membrane folds converging downward. Sometimes the entrance into the stricture may be distinctly perceived, even in the deepest area, occasionally, however, the folds so overlap that this is impossible.

A similar picture is sometimes presented to us by strictures due to caustics, and the previously mentioned protuberances with well-retained mucous membrane may also be produced by the bulging of the wall of the esophagus in consequence of compression from without. Therefore, the *proof of ulceration* or of a *cauliflower-like proliferation* entering into the lumen is of the greatest importance. Sometimes it is possible to recognize the line of demarcation where the healthy mucous membrane terminates, having an eroded appearance, or the transition to the ulcerating part, the surface of which can no longer be recognized, betrays itself here and there by free, floating shreds of mucous membrane. We may distinguish the ulcerated portion by the *dirty, greyish-yellow, purulent coating* under which and beside which the *greyish-red, uneven cancer surface* can sometimes only be brought to view by cleansing, and which is characterized by its *tendency to continuous capillary hemorrhages.* Sometimes after the introduction of the tube, blood oozes continuously from the ulcerated surface, and, in spite of careful cleansing with cotton, nothing more can be seen. In other cases foaming, *muco-purulent,* bloody fluid becomes visible, and with every expiration is forced up through the narrow passage with a gurgling murmur. If we are certain that no injury has been done with the esophagoscope at the point of lesion, the expulsion of hemorrhagico-purulent secretions from the stenosis confirms the diagnosis of carcinoma.

The pulsating motion of the esophagus, which is synchronous with the dilatation of the aorta, disturbs the investigation, particularly if the focus of the disease be situated at the point of bifurcation. But, with some practice, we become accustomed to this rhythmic motion of the picture. The respiratory displacement of the organ, by which its walls approximate one another in expiration, scarcely interferes, provided breathing is not too deep and rapid; it is *absent,* or almost so, when *the cancer has infiltrated the walls* of the organ; it may, therefore, be absent upon one side or in the entire circumference. On the other hand, there may be a disagreeable disturbance due to severe cough, which, in spite of cocain and morphin, is a common consequence of the existing laryngitis or trachitis. It is important that the patients be examined upon an empty stomach. If the field of vision is obscured by remains of food, such as portions of milk, particles of egg and the like upon the mucous membrane, and particularly over the narrowed areas of the rough cancerous surface, mistakes are readily made; in other cases it is easily possible to mistake these whitish masses for cancer.

A single inspection will not always suffice for us to recognize the

carcinoma. In cases in which the diagnosis is uncertain, small particles of tissue may be extracted and later microscopically examined, but great care is necessary in this procedure.

The esophagoscope should never be used immediately after sounding with a rigid tube. If the sound has been introduced into the esophagus to determine a stricture and to measure its distance from the teeth, even with careful manipulation the field of vision may be obscured with blood. It is well, therefore, to wait a few hours.

The results furnished by *auscultation of the esophagus* are in the main meager. If the patient takes fluid, swallow by swallow, he himself will frequently notice, as do those about him, *a gurgling sound* which is sometimes heard for a few seconds and is produced at the narrowed area by the checking and regurgitation of the fluid stream. By auscultation at the previously designated area, these *deglutition murmurs* give us diagnostic points of support, which, although not absolute proof, are nevertheless auxiliary, particularly in those cases in which, for example, upon suspicion of aneurysm, it is unwise to introduce instruments into the esophagus. No importance is to be attached to the rarity of the murmur in question (pressing-through murmur) or its absence; if it be present, however, and conspicuously prolonged, an impediment in the upper portion of the esophagus is indicated. *If, however, the pressing-through murmur is invariably absent,* simultaneously with the first sounds—an occasional absence is without importance—this is in favor of a pathologic process which limits the musculature in its function at the cardia, and which, according to experience, is, as a rule, of a carcinomatous nature; for only very exceptionally are both murmurs absent under normal conditions. Rarely does the esophageal bougie, even when it does not pass into the stomach, fill itself with fluid or pappy or compact remains of food which must have lodged in that portion above the carcinoma; they betray their origin by the absence of hydrochloric acid, pepsin, bile, and peptone, and by their alkaline or neutral reaction, which, however, may occasionally be acid, and is then due to organic fermentative acids.

The *gastric function* must suffer sooner or later, and earliest if the cancer be situated in the vicinity of the cardia. The hydrochloric acid secretion is therefore not rarely found to be absent. The *urine* is scant owing to the limited ingestion of fluid, and, in advanced stages, frequently shows the products of proteid decomposition (indican) and those of pathologic albumin decomposition (acetone, aceto-acetic acid, oxybutyric acid). Examination of *the larynx, lungs* and *pleura* should never be neglected. This often gives valuable aid in judging our cases.

DIAGNOSIS

The most important point in the **diagnosis of carcinoma of the esophagus** is the *recognition of a stenosis* of the esophagus. *If we determine this by the aid of the bougie in a previously healthy person, between the ages of forty and seventy, the affection having arisen without a recognizable cause, and steadily increased from weeks to months, nothing is more likely than that we are dealing with a carcinoma of the esophagus.* Yet a stenosis that has developed in this way, and under these conditions, may occasionally be of a non-malignant nature, and on account of the importance of this point for the patient, this possibility should first be excluded. It must be borne in mind that the history and the subjective symptoms, as well as most of the objective symptoms, may lead us astray. It sometimes happens, for I have myself observed and described such cases, that in benign dilatations and with the formation of a divertieulum as well as in spasm of the esophagus and atony, dysphagia may begin without assignable cause, and increase in a few months to a threatening degree, rapidly undermining the strength. On the other hand, in consequence of the chronic nature of disease of the esophagus, we must by no means permit ourselves to give a relatively favorable prognosis, and to exclude carcinoma. I have seen patients in whom difficulty in deglutition had existed from four to ten years prior to death, and due to carcinoma at the seat of the obstruction. These cases may be explained by the hypothesis that the cancer formed secondarily upon a base which had long been in a state of irritation, and had been well prepared. We know that ulcers due to a variety of causes (to tuberculosis, to caustics, or to digestive processes) in cicatrices, in inflammatory, irritative areas, therefore, wherever mechanical obstruction causes local irritation, prepare a soil suitable for the formation of cancer.

A second factor in the history, which can only be applied after thorough investigation, is the age of the patient. It is true cancer usually develops at an advanced age; but I have seen a number of cases of cancer of the esophagus in persons between 30 and 40 years of age, and hardly a year passes in which I do not see at least one case in early youth.

Another point which may mislead us is the *cachexia*. This is manifest in *all* diseases of the esophagus, provided they form a hindrance to the ingestion of food; in nervous disturbances and in dilatation of the esophagus, it may even be excessive.

It must be remembered that a *steady increase of dysphagia* for months, or even for years, occurs in various affections of the esophagus, namely, in simple inflammatory processes, in dilatation, and in diverticulum. A conspicuous *change* in the power of swallowing is present *in nervous disturbances* of the esophagus; but nervous influences may also occasionally appear in carcinoma.

Resistance to the passage of the esophageal sound is objective proof of importance in diagnosis, but this does not demonstrate with absolute certainty that there is an anatomical obstruction. *Spasm may arrest the bougie,* and it may be impossible to overcome this either by prolonged waiting or by attempts at swallowing on the part of the patient (see below). More important is the recognition of an *admixture of blood, of pus, or of the products of decomposition* which have lodged in the opening of the tube during probing, but it must be borne in mind that ulcerative processes and decomposition may be present in dilated portions of the organ without the presence of a carcinoma. Only when we are fortunate enough to find *shreds of tissue,* which unquestionably demonstrate the carcinomatous character of the affection, is this positive result a decisive proof; and this can only occur in exceptional cases; in the early stages of the disease when the diagnosis is still doubtful it rarely happens.

A *positive and early diagnosis* may be made most frequently by the *esophagoscope.* The esophagoscopic picture is generally the determining factor; in the majority of cases it may be readily obtained; in a minority this is unsatisfactory, i. e. it does not give a positive result; and this may be readily understood when we consider the fact that we are able to inspect only the upper border of the diseased area, and the later course of the affection and the consideration of all its accompanying circumstances can alone clear the situation.

From this it is evident that the positive determination of the character of a constriction of the esophagus is by no means easy. Although the malignant form predominates, many others must be considered which furnish a similar symptom-picture, and well deserve the interest of the physician. We should, therefore, avoid making a positive diagnosis of cancer of the esophagus, as is very commonly done, without certain indications such as dysphagia and resistance upon probing. Based on these criteria, no positive diagnosis is possible, and, when we consider how much is at stake for the patient, we should be somewhat more thorough in our differential diagnosis and more cautious in our prognosis.

We shall now describe the other manifold processes which cause obstruction to the passage of food, and, therefore, present the picture of stenosis of the esophagus. These are to be minutely discussed, and are best considered according to the classification given above. As benign neoplasms (myoma, fibroma, and cysts) are great rarities, and scarcely ever give rise to clinical symptoms, we may at once direct our attention to the *second group* of stenoses, those which originate from **inflammatory ulcerative processes.**

Stenoses of the lumen of the esophagus due to the *action of caustics* stand first in practical importance. These, as is well known, are not rare, and in regard to the symptoms it is immaterial whether acids, alkalies,

or other chemical agents, which have been swallowed either for purposes of poisoning or by accident, produce alterations in the mucous membrane. We shall not describe the stage of the acute, florid, inflammatory process and the phenomena to be attributed to this. We are more interested in the conditions which result in the course of weeks and months in consequence of infiltrations of the wall, formation of cicatrices, and narrowing of the lumen. The strictures which originate in this manner, if very narrow, annular or tubular, are limited to certain areas of the esophagus, and this is generally the upper portion, the region of the bifurcation, and the area of the foramen œsophageum. If we are dealing with long, tubular strictures, their centers are at these points, whence they distribute themselves upward or downward or in both directions. There may be strictures at *several points* of the esophagus, and by the coalescence of several, the constriction becomes extensive, and finally *total.*

The difficulties in deglutition are not specially characteristic. They are permanent, and vary in intensity according to the degree of the constriction and the local irritation, as is true of all stenoses. An aid in diagnosis is the circumstance that *from the history alone the nature of the disease may be determined with some degree of certainty.* But the anatomic condition of the organ caused by caustic we can only ascertain by an exact investigation, and here our first question is: Are we dealing with one or more strictures? In this respect, the reports of the patient regarding the act of deglutition permit some deductions. However, this information must be utilized with necessary reserve, as it is frequently shown to be erroneous. Moreover, we desire to know: Are inflammatory, ulcerative processes still present in the mucous membrane, or has definite cicatrization occurred in all parts? Is dilatation present? Finally, the question must be answered whether carcinoma has developed in consequence of the changes which are to be here considered. Information in regard to all these points is given us by *the sound* and *the esophagoscope.* Soft tubes, firm English and French bougies, also the metallic spirals which I preferably employ when it is necessary to find a passage, may be repeatedly used in order to acquaint ourselves with the situation and to enable us to form a correct opinion. The esophagoscope gives us accurate knowledge; strictures due to caustics form very characteristic pictures. In the neck and in the suprabifurcating area of the esophagus frequently striated, elongated, and flaky white cicatrices are outlined sharply against the rose-red mucous membrane; the nearer the stricture the more numerous the cicatrices upon the mucous membrane. The ring form, or the beginning of an annular stricture, manifests itself either as a cicatricial funnel at the apex of which a more or less circular or oval lumen is noted, or it resembles the portio vaginalis, from the fact that the region of the esophagus above it is dilated, and with the tube is uniformly forced downward above the invaginating stricture. Only with a very superficial cicatricial formation does the nar-

rowed area, whose lumen is often eccentrically layered from contraction of the cicatrix and frequently resembles a folded diaphragm, still show respiratory movement and radial folds; the deeper the effect of the caustic, the more rigid and immotile is the area of the stricture. The whole portion looks like a canal partly or entirely covered with white cicatricial tissue. At other times the mucous membrane up to the point of stricture is a deep, dark red, and shows a tendency to bleed readily; in the earlier stages of the disease, and even later, or as the result of improper treatment, we find ulcerative areas between the cicatrices.

If the caustic action has been slight, the secondary cicatricial formation may be of little extent, and after the acute symptoms have passed away no difficulty in deglutition remains; but in several cases of this kind I have observed that 10 to 20 years later difficulties in deglutition may reappear, particularly if the food is very coarse; larger, poorly masticated particles may be arrested. Thus, in adults, I have twice been obliged to extract coarse pieces of meat, about the size of a thumb-joint, which were arrested in the upper third of the esophagus. In both cases slight corrosive strictures were present which had been formed in earliest childhood. Up to that time the patients had ingested their food without any difficulty, and scarcely remembered the early disease of the esophagus. In both instances, the esophagoscope revealed a marked injection in that portion of the esophagus in which the bolus was arrested. Only in one of the patients was there a distinct cicatrix. With care further consequences were averted.

Stenoses due to *other ulcerative processes* are very rarely multiple. Mostly limited to a portion of the wall, the contracting cicatrix draws the corresponding portion of the retained mucous membrane toward it, and thus the stenoses cause *diverticuli and valve formation.* It may then happen that if the bougie is caught in the narrowing, it may appear of great extent, while, at other times, quite a large tube may be passed with ease. The differentiation of cicatricial strictures according to the nature of the ulcerative processes to which they are due is very difficult, and even impossible except in the case of the ulcer due to caustics. We consider chiefly *syphilitic, tuberculous, peptic,* and *diphtheritic* ulcerations. In doubtful cases the presence of syphilis and tuberculosis will aid in the diagnosis; peptic ulcers occur only in the lower third of the esophagus, and there are usually accompanying symptoms which point to gastric ulcer. I treated successfully a case of stricture of the esophagus following scarlatinal diphtheria. Naturally here also the preceding disease is a guide to the diagnosis. In conclusion we must consider cicatricial constrictions which have their origin in *peri-esophageal* processes which start from the lymph-glands, secondarily producing ulceration of the esophagus, which, in case healing occurs, results in stenosis of the lumen.

The finer differentiation of cicatricial strictures according to their genesis is not of practical importance. It is, however, always an advantage

to obtain a clear picture of the anatomic condition by aid of the esophagoscope, and thus determine whether or not florid inflammatory processes are present, and whether carcinoma, especially, can be excluded.

Of great import is the recognition of stenosis due to **spasm of the esophagus.** By the pathologic contraction of a layer of the annular fibers of the musculature, a segment of the esophagus becomes impermeable, or can only be passed with difficulty. The obstruction to deglutition resulting from this is of varying duration and intensity, and occasionally is also accompanied by painful sensations. The spasm may be transitory or permanent; sometimes the affection persists uninterruptedly for weeks or months or even for years, occasionally it is intermittent, and at other times only occurs after a definite irritation from food. In many cases this esophagismus *accompanies hysteria and neurasthenia,* epilepsy, chorea, and tetanus; it is an invariable and most important symptom of hydrophobia. Occasionally it is one of the manifestations of disease of the central nervous system; very frequently it is *reflex.* Thus, a spasm may prevent the ingress of our instrument, even of a soft tube; foreign bodies, coarse food, irritative fluids, anything that produces decided retching, may give rise to stubborn spasm. *Above all, spasm is frequently a concomitant symptom of other diseases of the esophagus.* Every anatomical process in the esophagus may be accompanied by spasm. Diseases of *other organs* may produce the spasm; affections of the pharynx and larynx, of the stomach and intestine, of the male and female genital organs, may be its etiologic basis, and *trauma,* which directly affects the head of the thorax, may cause reflex spasm. The appearance of spasm in certain *intoxications* (from belladonna, from strychnin, from sausage, etc.), following refrigeration with or without rheumatic affection of the joints and muscles, and with or without catarrhal affections of the larynx and pharynx, is worthy of mention.

This neurosis shows itself chiefly by dysphagia. The nature of the difficulty in deglutition is often not characteristic; the symptoms are the same as those complained of by patients with stenosis of the esophagus from other causes. Occasionally the spasm is accompanied by dull pressure in the chest, by a painful feeling of constriction, by lancinating or burning pains in the neighborhood of the esophagus, for example, in the shoulder or the neck, or it follows these symptoms. Spasm of the muscles of the pharynx, of the larynx, of the trunk, and of the extremity may coexist and alternate with esophagospasm, particularly in those cases in which a general neurosis is the foundation of the affection. The degree of hindrance in deglutition varies greatly. Only rarely is the esophagus absolutely, or almost wholly, impermeable for a long time; in these cases severe inanition may result, and the patient finally perish from the neurosis. This dysphagia may appear in dissimilar forms. Sometimes the patients experience only the sensation of an arrest of the food, and by the aid of a

few deep inspirations, or by swallowing some fluid, the bolus finally reaches the stomach; or the first food is regurgitated while that subsequently taken passes without difficulty. In some cases the spasm shows itself only upon abnormal irritation, for example, during probing or when certain foods are taken, usually coarse food or solid food in general; but we sometimes meet a patient who finds fluids more difficult to swallow than solid food. Occasionally the spasm occurs only at certain times of the day, or during the course of prolonged meals.

In spite of the fact that some of these disturbances we have mentioned certainly arise only under the influence of a derangement of innervation, positive proof that we are dealing only with a nervous affection can never be attained in the given case by the history and the subjective observation of the patient. *The diagnosis of spasm is rarely easy.* The most useful points of support for the recognition of the neurosis from the subjective and objective symptoms will now be briefly described.

The sudden appearance of difficulty in deglutition, particularly of fluid food, indicates the presence of spasm, and still more so the *intermittence of the attacks,* which may be quite irregular or be produced by very definite influences, partly nervous, the intensity and duration of which vary. *Differences in the degree of permeability* of the esophagus is a conspicuous symptom favoring spasm; the first food may be arrested, the succeeding may pass, or, the first masses of food may pass readily, and then, without assignable cause, a sudden stoppage occurs. More important is a *variation in the seat of the constriction,* so that, for example, on one day the upper, and upon another day the lower, third of the esophagus becomes impermeable. But this is a very rare symptom, and is only found as an accompaniment of severe nervous affections. An absolute diagnosis of this condition can only be made by means of *the bougie* and *the esophagoscope.* The first sounding should take place with a rigid instrument, as this will more certainly pass the axis of the organ than a soft one, which is easily bent. If we probe during a time when the difficulty in deglutition exists, a *resistance* must be objectively noted. After a short pause, or on moderate pressure, it may disappear, for there are many mild forms of spasm, but it *must positively be present.* It is worthy of note that the resistance may be caused to disappear by probing, by means of which we force the patient *repeatedly to swallow very rapidly,* and this brings about a more decided innervation of the longitudinal muscles of the esophagus, therefore, the dilators, and thus we overcome at least moderate grades of spasm. Moreover, probing may reveal a variation in the degree of permeability and in the seat of the constriction which is often of decisive import.

By means of the esophagoscope we are enabled to recognize *other esophageal diseases,* and *positively to diagnosticate spasm:* for this, as a rule, usually gives a characteristic picture,—sharply rising folds of mucous membrane which converge toward a point in the middle of the lumen and form

a rosette-like, more or less rigid closure. The lumen, if it can be recognized at all, is narrow. Only upon deep inspiration or on coughing are fluids (mucus, gastric contents) and air regurgitated through the narrow opening, not rarely accompanied by a hissing or gurgling murmur. The mucous membrane of the contracted portion of the esophagus shows abnormal, intense reddening. Occasionally the mucosa is found eroded, particularly in the region where regurgitation or stagnation has occurred.

If an examination be made during a time free from attacks, all the previously mentioned criteria may be absent, but a negative finding, in particular, is of diagnostic value. *The determination of the previously mentioned causes of spasm* is an important aid: *The proof of another affection of the esophagus coexisting, the recognition of a local affection of which the spasm of the esophagus may be the reflex symptom.* Above all, we must consider the status of the nervous system; but even if disease of this region be unquestionably present, it does not permit a conclusion regarding the character of a coincident difficulty in deglutition. This must be borne in mind if we would avoid falling into gross error. Inversely, we should observe that in true spasm also the difficulty in deglutition may progressively increase, that extreme cachexia may also develop, and that more or less complete impermeability of the esophagus may persist stubbornly for months and years.

Congenital stenoses of the esophagus, from the onset, either make life impossible, or they manifest themselves by a difficulty in deglutition which appears in early youth; it must be remarked here that congenital dilatation of the organ (anterior stomach) may give rise to symptoms similar to those of stenosis. If it is possible to demonstrate that considerable masses of food are retained in the esophagus, this is proof that dilatation at least is present; whether beneath this a constriction also exists can only be determined by the sound and the esophagoscope.

Occlusion stenoses, to the description of which we now turn, are due to impaction by *foreign bodies* which are arrested in the esophagus; they occlude the passage more or less completely, irritate the mucous membrane, and eventually may produce phlegmons and perforation. Even very small objects, for example, small fish bones, by giving rise to local irritation and spasm, may cause pain and symptoms of stenosis; and, even if the foreign body is not arrested but reaches the stomach, *erosions and fissures* which it produces in the mucous membrane may cause the same symptoms. On the contrary, it will be observed that not only small but also *comparatively large foreign bodies,* for example, a plate of false teeth, may be arrested in the esophagus *without giving rise to decided subjective, or even marked objective, symptoms.* For example, as I myself have seen, a plate of false teeth larger than a silver half-dollar may be arrested in the upper third

of the esophagus below the larynx, and lie with its convex surface in the concavity of the anterior wall of the esophagus, so that the organ gapes, and thus not only food but thick bougies may pass the large foreign body without difficulty. In this case, it had been supposed that the plate of false teeth was no longer in the esophagus, and the symptoms still present had been referred to lacerations in the mucous membrane; the true state of affairs was revealed by the esophagoscope.

Foreign bodies, almost without exception, are arrested in the *upper,* narrower parts of the esophagus; when they are found in the lower portions they have usually been forced down by instruments. With a previously existing stenosis even a small foreign body, for instance, a fruit kernel, may bring about complete obstruction of the esophagus, and absolute impermeability arises when a spastic process of the musculature holds a small foreign body.

If foreign bodies have been swallowed, the patient is generally aware of it, and reports the circumstance; where no *history* can be utilized, *the acute appearance* of stenosis at once leads us to think of obstruction; but also when the stenosis is due to foreign bodies, the *variation in permeability* occasionally favors this hypothesis. Temporarily the closure is complete, then the foreign body, in consequence of a paroxysm of coughing, or by retching, changes its position, and food now passes, perhaps also a bougie. But the sensation of a foreign body being lodged in the esophagus mostly remains, and after a short time difficulty in deglutition again becomes noticeable.

The examination with the bougie in doubtful cases will scarcely show the nature of the obstruction. By the introduction of instruments, we are at most only able to determine that a mechanical obstruction is present, and this is not even certain in all cases; for the probe may pass alongside of the foreign body through the lumen of the esophagus. On the other hand, *esophagoscopy always enables us to make a positive diagnosis.* By the aid of the eye we can extract the foreign body, or force it down, and readily cure any existing lesions in the mucous membrane by cauterization.

The **stenoses due to compression of the esophagus** are of the greatest practical importance. Any tumor formation in the neighborhood of the organ may sooner or later lead to compression of the tube. The esophagus is naturally motile, and may deviate. If, however, it is completely surrounded, or only adherent on one side, symptoms of stenosis appear. *Goiter* itself most readily causes difficulty in respiration, but rarely disturbs the function of deglutition; this will occur if it be very large, or if it surround the esophagus with greatly developed processes, or show malignant degeneration. Carcinoma of *the larynx* or of *the vertebral column* often interferes with the act of deglutition, and very early.

In the thoracic cavity, as well as in the neck, the esophagus may deviate

from compression. *Cancer of the lung, of the pleura,* or *of the vertebral column* must first proliferate into the peri-esophageal tissue before producing marked dysphagia. Enlargement of the tracheobronchial or mediastinal *lymph-glands* from cancer, tuberculosis, or syphilis more frequently disturbs the act of deglutition; as the result of purulent liquefaction of the glands, broncho-esophageal fistula may form, and later, upon cicatrization, true stenosis or traction diverticulum may develop. A massive *pericarditis* or a cor bovinum scarcely ever produces dysphagia, and only exceptionally will the pressure of an *aortic aneurysm* narrow the lumen of the esophagus. These patients more frequently complain of constriction and difficulty of respiration upon *swallowing* than of actual difficulty in forcing their food downward. The disturbances in deglutition from the presence of a *diverticulum of the esophagus* are due to various causes, and compression of the organ by the filled sac is only one of the most important, but, nevertheless, must be taken into consideration. The filled diverticulum may unquestionably completely occlude the passage into the stomach. If it is empty, food as well as the sound will often pass. This variation in the permeability has a certain diagnostic importance.

The differentiation between compression stenosis and other forms of narrowing of the esophagus is frequently not easy. Of utmost consequence here is the *differentiation of compression of the esophagus by disease of the neighboring organs from cancer of the esophagus.* In the esophagoscope the picture of compression stenosis *may resemble that which we observe where there are infiltrating tumors of the organ.* From one side a tumor with smooth, reddened mucous membrane protrudes into the lumen, and does not change its position upon respiration. The lumen of the organ deviates toward the side and is recognized as a small semilunar space, or it shows a funnel shape gradually tapering downward. This finding is not uniform; it may also occasionally be noticed in carcinoma. For the diagnosis of compression stenosis the *examination of the thorax* often reveals exact grounds of support; a retardation of one-half of the thorax upon respiration, the conspicuous appearance of veins upon the skin of the chest, dulness or abnormal pulsations, are important factors; expectoration, particularly if hemorrhagic, true hemoptysis, the signs of infiltration of the lung, exudation into the pleura, painful points upon the vertebral column, all aid us in the recognition of the underlying condition. Finally, in stenosis of the lumen by compression, the nature of the *dysphagia* and the *results of probing* may be characteristic. Occasionally, a crass *disproportion* may be determined *between* the *results of probing* and the *ability to swallow.* Such a disproportion is also found in atony and paralysis of the esophagus, in tumors developing in the walls of the organ, occasionally also when foreign bodies are present, and in hyperesthetic conditions, but in compression this symptom is of special import. While with atony and paralysis of the esophagus the sound invariably passes unarrested into the

stomach, in compression we note *resistance* whenever the instrument is passed; this is usually slight, and may be overcome with comparative ease by the use of moderate force; even a *very thick tube* may pass under these circumstances, while the patient is scarcely able to swallow thin fluid. The muscles lack the power to do what the pressure of the hand which guides the sound can achieve. Moreover, it is noteworthy that in these cases of compression *forced probing,* provided an aneurysm is not present, is not dangerous and is *well borne; traces of blood* are *scarcely ever noted* upon the bougie or in the fenestra of the tube, which, without exception, is always the case in carcinoma if the esophagus be energetically sounded. Where such a result of probing is combined with the esophagoscopic finding sketched above, carcinoma of the esophagus may be certainly excluded, for with a carcinomatous infiltration of the esophagus producing a prominent tumor, such as we have described above, the lumen of the organ must, under all circumstances, be decidedly narrowed, and we find it is impossible to pass a thick sound.

The differentiation from one another of the various kinds of stenosis which we have here separately analyzed is of the utmost practical value; it is naturally not always possible to make an exact diagnosis, but much is already gained if we are able positively to exclude carcinoma.

If it be doubtful whether cancer is present or not, and in my opinion this is frequently the case, in the interest of the patient all possibilities should be carefully investigated and treatment be directed accordingly. Thus, in the last few years, I have been able by energetic iodin treatment to keep alive two cases of suspected carcinoma of the esophagus, because I made a correct diagnosis of compression stenosis of the esophagus, and was right in assuming syphilitic enlargement of the glands to be the cause of the obstruction.

In many cases, even by the aid of the esophagoscope, it is impossible absolutely to define the difficulty, but this must not prevent us from making every effort to clear the complicated situation. Certainly the great majority of cases of stenosis of the esophagus are incurable, but, in the minority in which the affection is recognized, therapeutic success is possible, and, even if we do not cure the others, we can benefit them by treatment, and, by the clear insight which we have obtained of the character of the disturbance, can bring about a decided amelioration.

TREATMENT

In the *treatment* of all stenoses of the esophagus some *general therapeutic rules* are operative concerning *hygiene* and *dietetics.* As the total intake of food is frequently lessened on account of the difficulty of deglutition, such patients emaciate, even in cases in which there is no malignant process. Accordingly a leading and important object of treatment is to

produce a favorable influence upon nutrition. Nourishment should always be *plentiful,* and, under some circumstances, it should be calculated to increase flesh. It need scarcely be mentioned that in the solution of this problem great, frequently insurmountable, difficulties are opposed, yet even here much may be attained by the proper choice of food, and by the utilization of all auxiliary methods for artificial nutrition. We should make it a rule that the patients take *only such food as we know will pass easily through the narrow space;* for it is possible to take sufficient food even though it be in the thinnest of fluids. Trials with coarser food, in many cases, only increase the local irritation, and thereby make it more difficult to swallow liquid food. *Large particles may be arrested,* and thus render the stenosis completely impermeable; connective tissue shreds, in particular, which adhere to particles of fat and meat, obstruct with extraordinary ease a lumen which is already narrowed. For this reason we must exercise the greatest care to have the particles of meat, vegetable, etc., as small as possible, and where we are not certain that the patient masticates the food thoroughly with his teeth, these, so far as permitted, should only be given in a crushed form. That foods with skin, stems, kernels, and husks are forbidden is self-evident, but even *highly seasoned* or *spiced food* (mustard, etc.), concentrated alcohol, or very acid foods, are to be avoided on account of their irritative effect. The same is true of *extremes of temperature.* Lukewarm or slightly cooled fluids are easiest to swallow, and this does not contradict the fact that occasionally, where there is marked congestion and swelling, ice may be well borne; food of a low temperature is not suitable for prolonged use, as it unfavorably influences gastric activity.

Naturally, the *food in its composition* should contain *all the important nutritive elements,* and as we are able to administer not only salts and carbohydrates but even fats and proteids (the latter, for example, in the form of artificial foods) in a form soluble in water, it is quite possible to nourish the patient sufficiently in those cases in which only a thin fluid passes. I attach especial importance to the administration of *fat* in the form of melted butter, or olive oil, or cream. Apart from the high combustion value by which it is distinguished above all other food, it is particularly advisable because it makes the bolus soft and slippery, and because, adhering to the mucous membrane, it protects the same, thus diminishing the irritability of the inflamed parts; sometimes it will trickle through a stricture when even water is regurgitated.

The arrangement of meals and the manner in which they are eaten is of vital importance. All over-exertion of the diseased organ should be avoided. The patients are therefore advised to swallow *slowly, with pauses between;* where compact food is taken, the ingestion of thin fluid after each bite will facilitate its passage. Each meal, therefore, should consume a comparatively long time, even when only small amounts of food are eaten, but this does not matter; such patients should be encouraged to eat very

often. That mental rest and bodily relaxation are beneficial here is true, all the more so as unfavorable nervous influences very readily aggravate the disturbance in swallowing, even in those cases in which a carcinoma is present. The avoidance of all unnecessary exertion is absolutely essential for the purpose we have in view, namely, to improve the nutrition. Not rarely we observe that with complete rest in bed the power of swallowing improves. It need hardly be mentioned that everything that stimulates the appetite and increases the power of assimilation (fresh air, massage, wine, etc.) will aid in the treatment.

Further curative measures in patients with stenosis of the esophagus depend upon the nature of the underlying process. Let us first consider the treatment in the cases which are in the majority, in *carcinoma.* The object in cancer is this, to maintain the patient in such a condition that he is able to ingest fluid or pappy food readily, or, at least, without decided difficulty. We attempt to keep the stenosis from becoming impermeable, but it is not our aim to extend our therapeutic endeavors beyond the limit just mentioned, and to bring about greater ease in swallowing more compact food no matter at what cost. If it is clear to us to what the dysphagia in cancer of the esophagus is due, the two possibilities which are present here, and to which I have already called attention, will, above all else, prevent this. In the first place the disturbances in deglutition are produced by a *mechanical obstruction,* by infiltration of the walls, or by the proliferating neoplasm. The second factor of no less consequence is the damage to the *nervous* and *muscular apparatus* of the esophagus, which, in some of the cases, is in an extremely irritable condition, and reacts abnormally as soon as food reaches the ulcerated, inflamed area. This reaction frequently manifests itself as *spasm,* during which not a drop of fluid will pass the stricture, yet the same patient, after a preceding hypodermic injection of morphin, may readily swallow solid food. At other times *atonic* conditions react unfavorably upon deglutition in that portion above the cancer, and even in the areas in which there is no dilatation. From hyperesthesia of the inflamed parts, by over-exertion, etc., irregularity and weakness of function may arise and lead to an interruption, or, at least, to a disturbance of peristalsis which manifests itself by an unfavorable influence on the muscles, sometimes the longitudinal, at other times the annular, the final result, however, being the same, namely, a disturbance of deglutition.

If we observe these points, we must distinguish the treatment of mechanical obstruction from that of irritative conditions and atony. The former is a very unsuitable point toward which to direct our treatment; in general more harm is done here than good, yet all the more must we attempt to relieve the *local irritation and weakness.* Besides a bland diet, we must prohibit any food that does not pass easily, anything that may

cause regurgitation, and must consider an anesthetic process. *Morphin* administered internally is often beneficial; the objections to its prolonged use (becoming accustomed to the poison, and loss of appetite) are naturally not slight. Morphin may be administered alone, or in combination with cocain or antipyrin or menthol in the form of compressed tablets, which are slowly dissolved in the mouth; I use these very frequently in high-seated carcinomata which are near the larynx and pharynx, or which attack these organs (for example, morphin muriate 0.0025, cocain 0.0025, antipyrin 0.1, sacch. 0.3. M. f. tabl. compr. dos. 30; one tablet several times daily, immediately before eating).

Where morphin fails of success, I resort to *local anesthesia.* With the aid of the esophageal syringe devised by me (a simple syringe with a capacity of about 3 grams, and to which a fine tube of about 25 to 35 cm. in length is attached) I inject, reaching the point of the affection, a three to five per cent. *cucain solution,* at first twice daily, later less frequently: doses of from 2 to 3 grams of the solution per day are well borne and do not give rise to symptoms of intoxication. There are also cases in which cauterization of the hyperesthetic inflamed mucous membrane with a 1–3 per cent. silver nitrate solution may prove beneficial. The improvement is sometimes remarkable, even though, for the most part, only transitory; but, nevertheless, this process assists materially in attaining the object sketched above, namely, the retention of the patient's ability to swallow fluid and pappy food. The process of injection is quite simple, and may be employed by any physician; it can never do harm, but only good. The same may be said of mild measures to combat atonic and ectatic conditions —here, above all, mild *lavage of the esophagus* with the same instrument which we employ for the stomach is advisable and readily carried out: mucous masses, obstructing coagula, remains of food, foreign bodies, products of decomposition, etc., are thus removed, and, simultaneously, this cleansing process acts also as a kind of massage, and has a tonic effect upon the muscle.

All of these auxiliary measures are more worthy of trial in the treatment of the carcinoma than *probing,* by which the mechanical obstruction which causes the disturbance is increased. In my experience the majority of all malignant neoplasms are unsuitable for treatment by the sound, and only in a few cases have I been convinced of an improvement in deglutition by this process. *So long as the patients are able to swallow fluid and pappy food, I avoid every process which dilates mechanically.* In such cases we must be content with the existing condition, for the probability of attaining anything more satisfactory by sounding is very slight. If the ability to swallow is markedly disturbed, so that deglutition of thin fluids is only accomplished with extreme difficulty, if anesthetics do not relieve the condition, if lavage simultaneously is without result, we can no longer refrain from dilating the stricture. It must be borne in mind that sound-

ing is never an insignificant procedure; it increases the irritability of the organ, the tendency to hemorrhage and ulceration, and adds to the danger of perforation.

In probing, the hollow, fenestrated, hard rubber instruments may be used with advantage after being previously softened in warm water, but, nevertheless, by bending in the stricture, or even before they reach it, these are soon worn out. Solid bougies of the same material with a button-shaped or pointed end are more satisfactory, although they are not much more durable. I have for years frequently employed bougies which were manufactured for me after Crawcour's method. They are made from rolled sheet metal, cut into spirals, the flexible part, 40 cm. long, making up the coils; they terminate in a button-like attachment about 1½ cm. in length, the upper part consisting of a solid steel handle 10 cm. in length. They are very flexible, find their way into the stricture even if it is excentrically situated more readily than other sounds, and are almost indestructible if kept clean (a diluted lysol solution and subsequent drying by heat are sufficient for cleansing purposes). In carcinoma we must be extremely cautious in the passage of the sound, and never employ the slightest degree of force. The sound, which will barely pass, is allowed to remain for a few minutes; it should be introduced every other day, not oftener, for the diseased mass reacts to every irritation, bleeds readily, and thus edema and pain are rapidly produced. Often it is difficult to reach the narrow canal from the dilated portion above the stenosis, especially as this passage is not always straight; portions of the tumor may occlude the opening, or the sound may bend and be pressed against the wall of the organ, where even slight pressure may be followed by the most serious consequences. If the opening into the canal is not readily found, further manipulation should be stopped for a time; if, however, we succeed in passing an instrument of considerable size, at the next trial we should try most carefully to pass one a size larger. If we succeed in this, a still larger one may later be used. In the main, we must be careful to use no force in this method of treatment, but should rather be content with moderate success, and avoid too frequent and too prolonged soundings.

It will not rarely surprise us that although, for example, No. 5 passes readily, at the next attempt No. 2 can hardly be passed through the obstruction; acute swellings have appeared, or the tumor, in consequence of irritation, shows a more pronounced growth into the lumen of the canal. On the other hand, it may happen that the narrow passage will suddenly become permeable for compact food and thicker bougies owing to the removal of obstructions by ulcerative decomposition on the part of the tumor masses. If it is impossible to introduce a bougie in the usual manner through the narrowed esophagus, the attempt may be made with the patient in the recumbent posture, eventually *with the aid of* the *esophagoscope;* instead of working in the dark we may occasionally find the entrance to the canal

immediately, and we may sometimes succeed in cases where we have previously failed. For this purpose I employ spiral bougies (see above) or English bougies with a solid metallic guiding staff.

For dilatation of esophageal stenosis, still other instruments have been employed. Senator was the first to introduce *laminaria tents* of varying thickness into the carcinomatous stricture to attain a dilatation by their gradual swelling. The laminaria tent was fastened by a screw to a thin, flexible bougie. I have entirely abandoned this method in the treatment of carcinoma; but the idea may be well utilized in the dilatation of other strictures if the laminaria tents armed with a silk thread under the direction of the eye and by the aid of the esophagoscope are introduced into the narrow passage, and are permitted to remain from three to ten hours, perhaps even longer, and the tents are then drawn up by the silk threads. Dilatation may thus be promoted with extraordinary rapidity. We may soon convince ourselves that there is no danger in this process, and in desperate cases I have obtained from it excellent results (see below).

To produce a slow and careful dilatation, Schreiber advises a dilator consisting of an ordinary, thin, non-fenestrated stomach-tube, at the esophageal end of which a rubber tube 2 to 3 cm. in length is attached, which again terminates in a smooth firm point. At the opposite end of the sound there is a metallic attachment which is connected with a suitable syringe having a capacity of from 10 to 30 c.c. The sound is introduced through the narrow passage, water being injected into the sound so that the rubber piece fills and expands. The tense, elastic balloon we now attempt to withdraw through the narrow passage. This, in fact, is extremely difficult, but with great care quite decided pressure is exerted upon the internal walls of the stenosed area. This process I very rarely employ in carcinomatous strictures, as they are generally too unyielding to respond to the pressure. But the method is well adapted to soft, recent, cicatricial tissue, and to overcoming spastically stenosed areas.

[Professor B. W. Sippy, of Chicago, has invented an instrument for the dilatation of spasmodic and other strictures of the esophagus, especially cardiospasm (see Fig. 1). The instrument is especially applicable in hypertrophic stenosis of the cardia due to long standing cardiospasm. It has been successfully employed in several patients. In two patients under my charge the result was notable. In one, a woman with long standing cardiospasm and apparent hypertrophy of the circular fibers due to the spasm of the cardia, and with considerable dilatation of the esophagus, three applications of the instrument resulted in complete relief. Other methods failed after a trial of several months. The instrument is applicable in cases of organic stricture, but should then be used with extreme caution.—Ed.]

In conclusion I must mention the treatment with a *permanent cannula* which was so strongly advised in foreign countries, was tested by Leyden and Renvers, and has been of late especially praised by Curschmann. The

cannula employed for this purpose is best manufactured from elastic material, it should be 10 to 12 cm. in length, and have at its point a width

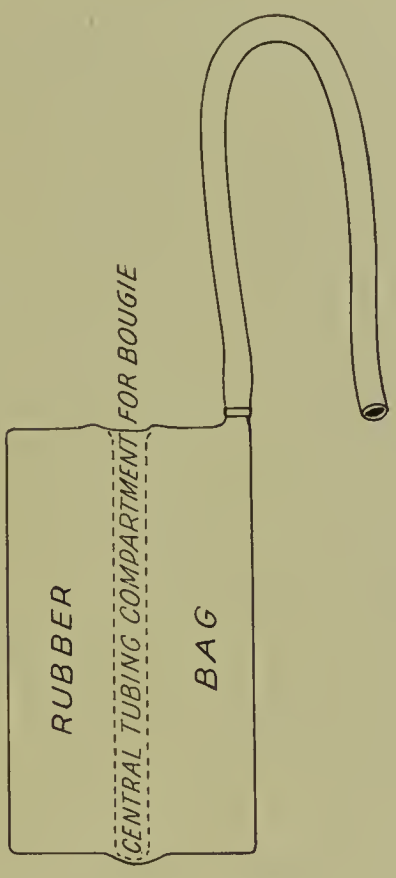

Fig. 1.—The Sippy Dilator.

[The dilator consists of a rubber bag 3 inches long and 1½ inches wide when collapsed. At an upper corner of the bag a firm piece of rubber tubing about 20 inches long is attached. Another piece of rubber tubing 3 inches long is secured in the center of the inside of the rubber bag. The lower end of this piece of tubing is closed; the upper end remains open and is secured air tight in the wall of the rubber bag. The tip of a whalebone bougie introduced into this compartment guides the bag into the seat of the stricture. The essential feature is a bag made of thin firm cloth which encloses the rubber bag, and limits accurately the degree of dilatation. The length of the cloth sac should be about 3 inches. Its width determines the degree of dilatation. In dilating strictures due to spasm the width of the cloth sac should be about 6 or 7 cm. for adults. Smaller sizes must be constructed for children and for dilating strictures due to malignant growths and cicatrix. The cloth sac must be uniform in width. When ready to introduce, a rubber condom is slipped on over the collapsed dilator and tied loosely about the rubber tubing and bougie. The distance of the stricture from the teeth is measured and the collapsed dilating bag guided well into it.

A firm rubber bulb such as accompanies an ordinary Paquelin cautery may be used as an air-pump. The dilating force that may be applied directly to the stricture is enormous, but limited accurately by the size of the cloth sac.—Ed.]

and lumen of about 5 mm. and above of about 12 mm. Such a cannula is introduced by means of a whalebone stylet with two ivory buttons; the lower button serves as an obdurator. The upper is larger, and permits

the introduction of a tube which is held by means of a silk thread. After the stylet has been withdrawn the thread is fastened upon the cheek. The advantage of this process is that nourishment, either fluid or pappy food, may be introduced in quite large amounts without difficulty. It is also said to prevent the decomposition of the cancer, as the food does not come in contact with the tumor. My experience with the use of permanent cannulæ is not particularly favorable. The retention of the silk thread in the mouth is uncomfortable to the patient; if it is drawn through the nose, it irritates the epiglottis and the pharynx, and is most unpleasant to some patients, for example, if catarrh of the larynx or of the pharynx, which is so frequent, is present. The mechanical irritation of the carcinoma is also not to be underestimated. Encircling of the tube by its growth, and clogging of the same, are not rare. The most unpleasant feature, however, is this, that occasionally in withdrawing the cannula the thread breaks, and we are then compelled to remove it with the esophagoscope, and this may be a very difficult operation. Above all it must be remembered that this cannula treatment is only adapted to patients in whom a tube of from 5 to 6 mm. still passes the obstruction. There are, however, patients in whom for some time sufficient nourishment may be introduced according to the method I have first described, without subjecting them to particular discomfort. The methods with laminaria tents and with the rubber ball sound presuppose a certain permeability of the stricture, although not to the extent necessary for the cannula, but this is evidently one of the reasons why these methods have not been more frequently resorted to in the treatment of carcinoma.

If the stricture is too narrow, so that probing with and without the esophagoscope gives no results, and the local application of drugs to combat the hyperesthesia and the spasms affords no material relief, when, therefore, the condition is such that, in spite of all endeavors, no fluids, or only slight amounts, trickle through the esophagus, and the patients rapidly emaciate, in my opinion the time has arrived for *surgical interference*. But before deciding upon this, I would urgently advise the simple trial of putting the patient to bed for a few days, with absolute rest, and to nourish him by the bowel, entirely refraining from the introduction of food by the mouth. Hunger and thirst may be allayed by small doses of cocain (0.02 per dose, two or three times daily), and the mouth and pharynx should be frequently rinsed with ice water. A favorable change is often noted in that the local condition improves decidedly. After these few days of rest, the passage is likely to be decidedly more free than it formerly was, and the introduction of the tube or the injections may be resumed should this appear advantageous; sometimes it is unnecessary, for we find that if such patients are nourished with enemata on one or two days in the week they are able to swallow quite well during the remaining period, particularly if small doses of morphin are simultaneously administered. In this

stage of the disease we have little hope of long averting the catastrophe, but whether in this crisis gastrostomy will greatly avail appears to be very questionable.

In my opinion, only in the most unfavorable cases is the production of a gastric fistula indicated. This operation is, in truth, for me the *ultima ratio,* notwithstanding the fact that the operation, through the successes of prominent surgeons, has become a relatively safe one, and that the fistula functionates perfectly for a long time. *Gastrostomy* appears to be absolutely indicated when *broncho-esophageal fistulæ* cause the continuous passage of food into the wrong channel, when imperfect permeability of the esophagus for any kind of food exists, and, finally, when in an early stage of the disease impassibility occurs and causes a rapid loss of strength. In well-advanced cases, with decided stenosis of the lumen, a marked prolongation of life, a noticeable improvement in the general nutritive condition, and a decided amelioration of the dysphagia is not to be expected from the operation. As a prophylactic measure there is not the slightest reason for early operation while there is still sufficient permeability of the esophagus. The *radical treatment* of cancer of the esophagus by extirpation could only come into question with high-seated tumors; up to this time not a single case of recovery has been reported. This is partly due to the great difficulties which the surgeon must combat, but partly also to the fact that the diagnosis is not made early enough. The more thorough use of the esophagoscope may, perhaps, be productive of better results in the near future.

Of other *symptomatic treatment,* I shall only mention the irrigation of the esophagus with disinfectant solutions (thymol 0.5–1000, salicylic acid 1–1000, silver nitrate 1–1000, etc.) to remove the symptoms due to stagnation (fetid odor, eructations). For the catarrh of the pharynx, of the larynx, and of the bronchi, which so frequently accompany the condition, I employ Ems water, morphin, etc.; the salts of iodin, also, bring about amelioration by thinning the tough mucus. The many neuralgic pains are relieved by our reliable antirheumatics.

The therapy of *cicatricial* strictures must be constructed on principles entirely different from those that come into question in carcinoma. Here there is a possibility of preserving life, under favorable circumstances of permanently curing the local lesion, and here *dilatation of the stricture* is the most important aim of the treatment. The results depend very much upon the methods of procedure. We shall first describe the simplest process, that of *sounding.* It is generally admitted that *slight* pressure is permissible in shoving the instrument through the narrowed cicatrized area, and that it may be permitted to remain for some time (from three to fifteen minutes). Daily probing is not always advisable; often a certain amount of forbearance gives better results, so that the method is only employed

every second day. If we are dealing with an excentrically layered canal, or with a somewhat circular narrow pass, the metallic spiral probes such as I advised many years ago, appear to me to be the most suitable instruments. If the way into the stricture may be found more easily, it is quite immaterial of what the dilator is made. Sounds with a button of whalebone, or English or French bougies, may be employed. If it is impossible to attain our end by the usual sightless method, before we conclude upon further surgical procedures (esophagotomy, gastrostomy) we should always make an attempt to probe with the aid of the esophagoscope. In apparently quite desperate cases I have at last succeeded in finding the entrance to the stricture, and in passing it with a fine instrument. The process is laborious, but the results are sometimes brilliant. For dilatation with the esophagoscope, I am partial to the use of metallic spiral sounds, or so-called "director sounds," i. e., narrow bougies of impregnated tissue which terminate in a quite long metal staff that permits of firm direction. Occasionally I have also employed with advantage fine lead and zinc staffs to direct the passage through the obliterated organ. After finding the way a few times with the esophagoscope, I frequently succeed in attaining my purpose upon simple probing in the recumbent posture without the introduction of the esophageal tube, and if the canal has been at all dilated I resume the trial in the sitting posture with the ordinary sound. A number of little artifices, which I do not care to describe here, may facilitate this process in difficult cases.

If the cicatricial tissue is not too firm, and the narrowed pass not too long, I find Schreiber's sound, cautiously used (see above), particularly useful in producing dilatation with comparative rapidity.

The treatment of cicatricial strictures by bougies should be begun as early as possible. This treatment should be carried out every day, and should be continued for a long time, for, even after extensive stretching, the tendency of cicatricial tissue to contract continues. Although we may attempt decidedly more in this affection of the organ than in carcinoma, cautious manipulation is nevertheless necessary, and it must be borne in mind that, besides cicatrices, ulceration may also be present, that the mucous membrane above the narrowing is frequently irritated and inflamed, so that here, as well as in cancer, the disturbance in deglutition does not necessarily depend upon the mechanical obstruction alone, but may be due also to nervous disturbance, to a tendency to spasm, and to atony.

There are, however, cases where the most persistent use of the bougie, even if it really pass through the constricted area, produces no noteworthy dilatation; and we often meet with cases in which it is impossible to pass the stricture with any of our instruments. In difficult cases of this kind, and sometimes in almost desperate situations, an attempt must be made to dilate in the esophagoscope with sounds or by the introduction of laminaria

tents at the starting point of the stricture. The latter process, which Ebstein has advised as a modification of the earlier Senator method (which see), I have repeatedly tested. For the introduction of the stylets into the stricture I employ forceps with smooth, concave, internal surfaces; if the entrance to the stricture is extremely small, the lower end of the tent is pointed, and cautiously pressed into the narrow canal. During this treatment the patient must be very carefully watched. At first the tent should be allowed to remain only for three to eight hours, provided there is no pain or fever, and later, for a longer time. Withdrawal is usually easy by the aid of the silk thread. In elongated strictures the process with the laminaria tent is an arduous one, and gives only very slow results. In these cases we should always try to attain our end by the introduction of long thin bougies by the aid of the esophagoscope. After we have found a passage, according to v. Hacker and Ebstein, dilatation may be facilitated under some circumstances by employing drains which are placed over a thin metallic introduction rod of about 2 mm. in size; if the staff is withdrawn the tension lessens, the drain contracts, and thus exerts decided pressure upon the wall, particularly if it is allowed to remain for a longer time, up to 24 hours. In my own experience the process is somewhat painful and not absolutely harmless; even with close watching of the patient, the jerking back of the rubber may occasionally cause a laceration in the wall of the organ.

If we do not succeed by these methods, if the results are unsatisfactory in that deglutition is not facilitated, and the patient's nutrition now suffers, *gastrostomy* should not be too long delayed. By producing a gastric fistula, we need no longer be anxious about the nutrition, we may influence favorably the irritation and inflammatory condition created in the esophagus by the inactivity of the organ, and may devote ourselves to the object of restoring its permeability. For this time is often necessary, much time. Besides, there is the added advantage that through the wound produced by gastrostomy we may attack the focus of the disease in the esophagus. Primarily this is possible by means of bougies, and cases have been reported where dilatation has been produced from below at a point which could not be reached by sounding through the mouth. This presupposes that the fistula is located in such an area that from it we may with comparative ease reach the cardia. This, unfortunately, is not the case with the fistulas commonly produced by Witzel's method; their advantage consists in their permitting an excellent occlusion, but manipulation with sounds from the gastric opening is exceedingly difficult. There are methods, however, which, even under these circumstances, may be quite well employed for dilatation. The simple experiment, after Socin, with *silver balls* which are fastened to a silk thread should first be tried. A very small one may be swallowed by the patient in the evening, and we are sometimes surprised to find that in the course of the night it has found its way into the stomach. Various

processes have been devised for securing through the fistula the silver thread which has been fastened above through the cheek. The stomach may be filled with water or soup, and these fluids are then permitted rapidly to flow off through the tube: not rarely the thread will be carried with them. Or slightly curved instruments (dressing forceps) must be introduced into the stomach and an attempt be made to catch the thread while the patient assumes different positions, and to draw it into the fistulous canal. If this is successful the ends of the silk thread are fastened together, the ring is closed, a fine rubber drain is inserted within it, and an effort is made to draw this through the stricture. Then larger drains may be employed, and the stricture be thus dilated. But we must not be too hasty, and too forcible pulling upon the thread must be avoided, as this causes great tension in the narrow pass, and the danger of injury is not inconsiderable.

It must be mentioned that *esophagotomy* may also be occasionally employed if it can be carried out *below the obstruction.* Nutrition is possible through the fistula, and, under some circumstances, dilatation may be attempted at the opening in the neck. Here, however, it must be observed that a stricture in the opening at the neck of the esophagus may be diagnosticated positively, but it is by no means certain that there are not also other impermeable ones in the lower part of the organ. Then we have to deal with a new obstruction. This we may occasionally remove from the wound in the neck by probing, or cutting, but such a process is always protracted, and, in the meantime, the exhausted patient may perish. Therefore in all doubtful and difficult cases it is wise to assure ourselves of the nutrition by the production of a gastric fistula. In the treatment of *compression stenoses,* sounding is also useful, and generally still more so than in cicatricial constriction, for we may manipulate the organ to a greater extent, the mucous membrane being everywhere intact.

We are soon convinced that a decided improvement in deglutition is not to be brought about by bougies; but if, at regular intervals, we introduce sounds with fenestrated openings into the stomach the nutrition of the patient may be carried on satisfactorily, and thus inanition be prevented. This is all that we can really accomplish in patients with compression stenosis, if the treatment does not diminish the size of the tumor which causes compression or remove it. The latter, unfortunately, is only exceptionally possible; for example, in syphilitic lymphomata by potassium iodid or an inunction treatment, in diverticulum by cleansing or extirpation of the sac.

In spastic contracture of the esophagus, soundings may occasionally be of use; I have frequently brought about a lessened tendency to spasm at the cardia by over-distention of the esophageal orifice of the stomach, for which Schreiber's rubber balloon sound serves the purpose best. This instrument is carefully introduced into the cardia, perhaps by the aid of several attempts at deglutition, or by local anesthesia, when the balloon is

distended, and pulled through the eardia. Above all, however, we must endeavor to ascertain the cause of the spasm, whether inside or outside of the esophagus, whether produced by local disease, or whether the general condition produces the tendency to spasm. The etiological therapy which results from this is of vital importanee, but, for this reason, the importance of the local symptomatic treatment is by no means to be undervalued. Besides sounding, the injection of remedies that produce anesthesia (eucain, 3 per cent.) or electricity by the aid of a simple esophageal electrode, such as I have proposed, may be of decided benefit. Where the spasm, particularly at the cardia, has produced secondary changes (dilatation, inflammation), the resulting eonditions are naturally to be also eonsidered in treatment.

In conclusion, a few remarks may be in order coneerning the *treatment of obstruction stenosis;* polypus (an extraordinarily rare occurrence) should be removed surgically; in obstruction due to masses of thrush, irrigations and the use of borax (3 per cent. solution, a tablespoonful every two hours) internally, are very serviceable. When *foreign bodies* are lodged in the esophagus, we must above all things refrain from at once employing the sound. We must first endeavor to remove the object which has entered the esophagus by the mouth, particularly if it has pointed or sharp edges, for, if forced downward, these may give rise to dangerous symptoms in the stomach and in the intestines. Smooth bodies, for example, coins, even if large, are not very dangerous in their journey through the digestive apparatus. It is, however, of the utmost importance that (if possible) a foreign body be speedily removed, provided we may extract it under the direction of the eye. Esophagoscopy enables us to achieve this in many cases by a bloodless process, where formerly an operation was neeessary; small pointed objects, particularly, are thus rapidly removed and without danger. Instead of the manifold instruments which were formerly neeessary (coin eatchers, hooks, esophageal probangs) we now, in most cases when employing the esophagoscope, use only a simple foreeps. Combined with this an instrument constructed aecording to the principles of the Leroy curette may sometimes be useful. That a small foreign body which causes disturbance of deglutition may sometimes be removed by the administration of an emetic must be also mentioned. *Fissures and erosions* which subsequently cause difficulty are best treated by the application of caustie under the direction of the eye, provided rest of the organ and morphin do not bring about rapid reeovery.

If the foreign body reaehes the stomaeh, the old effieaeious potato treatment for its expulsion per anum is still the best method: For three or four days the patient is given daily 3 or 4 pounds of potatoes prepared in different ways, constipation being induced by the administration of the tincture of opium; then the bowel is cleansed by enemata or the administration of castor oil.

THE HISTORY AND CLINICAL INDICATIONS OF GASTRIC LAVAGE

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At the turning point of the century the treatment of gastric diseases has reached a high degree of development. This progress has taken place upon the fruitful soil of an exact method of diagnosis based upon precise indications, and the seed was the ingenious idea of Kussmaul promulgated about a lifetime ago: Introduce a tube into the stomach, remove the abnormal contents by washing, and treat locally the diseased gastric mucous membrane.

Up to the middle of the previous century, but few of the pathologic changes of the stomach could be recognized with certainty, not even those for which pathologic anatomy had already furnished an explanation. Our knowledge of functional disturbances of the stomach was even more obscure. Digestive disturbances whose organic foundation was unrecognizable were attributed, according to the views of the time, to pathologic processes of the stomach, and were thus treated.

Naturally empiric treatment of an assumed affection could only produce unsatisfactory results—such as we sometimes attain even to-day when a greatly eulogized remedy is used to relieve a prominent symptom in a clinical case.

The modern physician who has been trained to employ his therapeutics according to definite indications and with clear ideas possesses also, without effort on his part, an advanced technic which is the fruit of the labor of his predecessors; he can scarcely realize the conditions in that time when modern auxiliary measures did not exist, yet in these we have not advanced to the fullest extent. And he will correctly measure and fully appreciate the value of his inheritance only when he studies its historical development and considers the labor that has brought to completion so great a work.

THE HISTORY OF GASTRIC LAVAGE

At the close of the sixth decade of the preceding century, dilatation of the stomach, particularly that advanced form developing from narrowing and closure of the pylorus, was reckoned among the diseases most difficult to treat. "Only exceptionally," says Kussmaul, "did we attain any results

in the treatment of this distressing affection. As a rule, little could be done to ameliorate the sufferings, and nothing to cure."

Cases of this kind were looked upon as hopeless, and for this reason were not welcomed in hospitals and clinics. Thus Kussmaul, at that time Director of the Freiburg Medical Clinic, was disinclined to admit to his Clinic, which was then greatly overfilled, a country girl, Marie Weiner, from Heimbach, aged 25, who sought admittance upon April 15, 1867. She had been suffering from a gastric affection since her eleventh year, having an extreme dilatation of the stomach due to pyloric ulcer, hypertrophy of the pylorus, and chronic catarrh of the stomach; she was greatly emaciated and suffered besides from peculiar spasmodic attacks which are now known and dreaded under the name of gastric tetany; however, at the repeated urgent entreaty of the patient, Kussmaul relented and admitted her to the Clinic.

This patient suffered almost daily from gastric pain and vomiting, and passed sleepless nights of suffering unless relieved by the administration of morphin; her pitiful condition gave Kussmaul the idea of employing the stomach-pump. His considerations and reasons were as follows:

"Quite frequently, when I observed the patient in the miserable prodromal stage of vomiting, the thought had occurred to me that I might ameliorate her sufferings by the employment of the stomach-pump, as the removal of large masses of decomposed acid gastric contents should cause the agonizing burning and retching at once to cease. The introduction of the esophageal sound was naturally not difficult, for where a gastric dilatation has existed for so long a time the esophagus also is usually dilated. The artificial emptying of the stomach by the pump could be no more painful or distressing than her condition before and during vomiting. At all events, it would be more rapid and complete than the natural emptying of the stomach by the act of vomiting, with its prolonged prodromal stage of nausea, pain and retching. Repeatedly, even after vomiting, palpation and percussion revealed that the stomach still contained considerable masses. This condition reminded me of the so-called ischuria paradoxa, in which large amounts of urine flow daily from the dilated urinary bladder without its actually becoming empty and without reducing its circumference. By means of the pump we must succeed in emptying the stomach *completely*, and, if its elastic and contractile power have not been entirely exhausted, perhaps even *give to it the tone to contract to its smallest extent*, just as the catheter occasionally brings about recovery in ischuria paradoxa.

"In our patient the gastric dilatation was due to constriction of the pylorus. At the autopsy of cases of extreme gastric dilatation I had repeatedly observed that the stenosis which caused them would still permit the passage of a small finger from the stomach into the duodenum, although toward the end of life there had appeared to be complete closure of the pylorus. In such cases I had occasionally noted at the bedside through

the abdominal covers active movement in the stomach. A paresis of the gastric musculature might be present, but certainly not complete paralysis. It appeared to me *as though the excessive distention, the filling and overloading of the stomach itself, produced a mechanical action which increased the constriction of the pylorus to complete closure,* and this condition I hoped to remove by emptying the stomach and decreasing its size.

"Finally, it appeared that the employment of the stomach-pump would permit a *more active topical treatment of the diseased gastric mucous membrane* than was formerly possible. In the case of our patient this organ had for years been continuously irritated by extremely acid contents. The retention, stagnation, and decomposition of masses of food in the stomach because of pyloric stenosis is certainly often the only cause of the catarrh of the mucous membrane; as, for example, in cases in which originally there was but an ulcer or a cicatrix at the pylorus, the stomach being otherwise intact; in all other cases it probably maintains and increases a catarrh already present. This is probably why we note so constantly, in constriction of the pylorus, that the mucous membrane about the pylorus, where the gastric contents especially accumulate, shows most intense disease. The stomach-pump, I hoped, would not only make it possible completely to evacuate these acid, irritating masses, but would also permit the washing and cleansing of the diseased mucous membrane which had been irritated by acid and alkaline fluids, as, for instance, with Vichy water or with an artificial soda solution."

In pursuance of these considerations Kussmaul, upon July 22, 1867, for the first time, pumped and washed out the stomach of his patient.

"The introduction of the stomach-tube, the pumping out, and the washing with Vichy water were unexpectedly easy. We withdrew three liters of acid, dirty-grey, sarcina-containing fluid, with particles of food of all kinds undergoing softening and decomposition.

"Even the immediate result of the first emptying and washing of the stomach with Vichy water was a surprisingly beneficial one. The patient, who was previously always exceedingly disagreeable, and of such a whining disposition that she well bore out her name, 'Weiner' (crier), appeared a few hours later as if completely transformed. For the first time she was agreeable and appeared comfortable in her bed, and she declared that for years she had not been in such good spirits. She at once digested and slept much better, and for two days was entirely free from depressing sensations in the stomach. After repeated employment of the pump, at the end of fourteen days the patient had a more healthy appearance, and had become another being. She who had always been disagreeable, had lain in bed or reclined in an arm-chair, was up the whole day, was very friendly, and attempted to make herself useful about the room, and soon in other parts of the house. In the first three months of her stay in the hospital, and before the employment of the pump, she had gained at most about 5½

pounds in weight; two months afterward she had gained at least 16½ pounds, and in not quite six months, 24 to 25 pounds. For two years recovery has been complete, although the patient is by no means in easy circumstances."

The first communication regarding this *new method of treatment of gastric dilatation* was reported by Kussmaul at the first meeting of the Section of Internal Medicine of the Forty-first Congress of German Naturalists and Physicians in Frankfort-on-the-Main in September, 1867. Further observations and experience regarding this treatment, which, in the meantime, had become a method of gastric dilatation, were communicated by Kussmaul in his Freiburg pro-rector oration on the 9th of September, 1869, and in his classic treatise in the *Deutsches Archiv für Klinische Medicin*, vol. vi, 1869, which has become widely celebrated: "On a New Method of Treatment of Gastric Dilatation by means of the Stomach Pump."

Besides the new method of treatment, this epoch-making work of Kussmaul's furnished such an enormous amount of clinical material and so many new points of view for pathology, diagnosis and treatment that it became the foundation, and even a treasury, for most of the later investigations in gastric disease. Besides benign constriction of the pylorus from ulcers, cicatrices, hypertrophy, and malignant constrictions from cancer, here was found the complete clinical investigation of the simple dilatations of the stomach not dependent upon stenosis of the pylorus or of the duodenum, those due to *atony of the muscularis* in consequence of too great weight and extension of the stomach beyond its elasticity from polyphagia, or paralytic weakness in convalescence from exhausting diseases, for example, enteric fever, or which had been produced in nervous anemic conditions, as well as *paresis* of the gastric musculature in consequence of *fatty and choloid degeneration of the muscular fibers.* The definite *mechanical factors* were also considered which occasionally during life had caused symptoms of complete closure of the pylorus while in the cadaver a small finger could readily be passed through the narrowed pylorus into the duodenum. The *descent* in gastric dilatation of the *pyloric portion of the stomach* which is most bulged out and implicated, and the formation of the *fetal* (vertical) *position* of the stomach were also discussed, as well as the possibility of a reflex *tonic spasm of the hypertrophied pylorus,* which is manifest to a decided degree in consequence of the irritation of the sensory nerves of the pyloric region produced by the acid contents which cause increased peristalsis in this neighborhood.

That this increased irritation of the gastric mucous membrane from stagnating gastric contents produces reflexly an *increased,* even *continuous* secretion of fluid, and not only of HCl-containing gastric juice, was at that time unknown to Kussmaul. It did not, however, escape the sharp eye of the investigator that in his patient suffering from tonic muscular spasm the amounts of fluid ejected by vomiting and brought up with the

stomach-pump were much greater than those introduced; he also noted the decidedly diminished amounts of urine in such patients. Kussmaul believed for a long time, as I know from his personal communications, in a kind of transudation into the stomach, but he was unable to furnish exact clinical proof of such an influx of fluids; as is well known, this experimental success was achieved only a few years ago (1893) by v. Mering. On the other hand, the significance of the marked, and often rapidly occurring, losses of water in some cases of gastric dilatation was quite clear to Kussmaul; he considered the tonic muscular spasms which were first described by him (so-called gastric tetany) to be the *consequences of rapid inspissation of the blood and a drying of the nerve and muscle,* and consequently, at that time, he laid great stress upon *the introduction of fluid in the form of meat, broth, and wine enemata,* which were absorbed in the intestine with beneficial effect. As an additional therapeutic resource which we obtained simultaneously with the stomach-pump, we must mention Kussmaul's experience that the dilated stomach which is no longer capable of retaining large amounts of fluid or food is still able to force into the intestine *small portions given at stated intervals,* and, finally, the employment of the *hypogastric bandage* to prop up the descended and dilated stomach.

Kussmaul, in his classic work, refers at different points to the variation of the stagnant contents of dilated stomachs in regard to amount, and to the appearance, the composition, and the degeneration of the gastric juice, in such a manner as to sound like a challenge to investigators to attack these questions. In truth, all of the clinical laboratories, particularly in Kussmaul's Clinic, were actively at work on this line, but were interrupted by the Franco-Prussian war and, later, by the great clinician's removal from Freiburg to Strassburg. The contents of diseased stomachs and, later, those also of healthy stomachs, were subjected to chemical. physiologic, and subsequently also to bacteriologic, investigations. Normal and pathologic processes of digestion attained more and more prominence, both from a clinical and a professional standpoint. With an eagerness without parallel. the tube was introduced into the stomach and gastric contents were brought up as if buried treasures were being restored to daylight.

Thanks to these eager investigators, we gradually became aware of the importance in diagnosis and prognosis of *the presence or absence of hydrochloric acid,* not only in gastric dilatation but also in gastric affections in general. *Simple color reactions* were found which, even at the bedside, showed the qualitative presence of hydrochloric acid and of organic acids, while analytical methods by measure and weight were formulated in great numbers to determine quantitatively the gastric acids. In connection with physiologic experiments in digestion to show the activity of the enzymes of the gastric juice, and their presence or absence, these quantitative analyses also made it possible to test the function of secretion of the gastric mucous

membrane and the digestive power of the gastric juice. We learned to recognize an *amylolytic and a proteolytic stage of gastric digestion, also the difference between combined and free hydrochloric acid.* Gradually a period dawned in which we believed that the activity of the stomach and the degree of its digestive disturbance could be definitely measured according to the amount of hydrochloric acid present, and the other functions of the stomach, especially its motor activity, were not sufficiently appreciated. A reaction took place. In the meantime, however, the number of methods for estimating free and combined hydrochloric acid became enormous, and the literature of this subject has become so extensive that it can scarcely be reviewed; the future will determine what portion of this is necessary, useful, and of permanent value.

But, to return to our theme of gastrie lavage, we must first mention *the instrument* which Kussmaul used to empty and wash out the stomach.

Kussmaul first employed the stomach-pump of the American physician, Wyman, which, according to Bowditch's reports (1852), was also utilized in draining empyemata. Then, however, he had pumps manufactured by the instrument maker, Fischer, in Freiburg, after Wyman's drawings, and these were also made use of by Reich in Stuttgart who designated them as Fischer's stomach-pump, without any mention of Kussmaul's authorship.

v. Ziemssen later ascertained that the honor of inventing this very useful instrument belongs neither to Wyman nor to Bowditch, but to a German instrument maker by the name of Weiss, who lived in London in the second decade of the last century. Ziemssen therefore proposed in 1870 that "the instrument which, by Kussmaul's brilliant discovery, rapidly attained such great distinction in the treatment of gastric diseases, should hereafter be designated as *the Weiss stomach-pump.*" At first Kussmaul, after once emptying and cleansing a dilated stomach, only employed the gastric pump when new difficulties arose, such as burning in the stomach, acid eructation, pain, a tendency to vomit and similar symptoms.

In consequence, the *daily employment* of the stomach-pump, particularly in severe cases, became more and more obvious, and it was also ascertained that for *emptying and washing the dilated stomach, the time when the stomach was still empty, therefore, the early morning,* was the best time.

As long as the patients remained in the hospital, this operation could be easily and regularly carried out early in the morning upon an empty stomach. After discharge from the hospital, however, the patients are unfortunate, "for a busy physician cannot find time to use the pumps for such patients for weeks or even for many months with the desirable regularity." These reasons induced Kussmaul to *teach the patients themselves to introduce the stomach-tube, and to wash out their stomachs.* His patients who had learned to introduce the stomach-tube and the stomach-pump for themselves—as the clinical histories of the year 1869 show—left the Freiburg Clinic with these instruments in their possession. It is very

interesting to note the use which a patient of Kussmaul's, a wool-spinner by occupation, made of the stomach-pump as soon as indigestion appeared. This patient by no means adhered to the directions which he had received in the Clinic, but, nevertheless, he improved steadily. If the foods which were forbidden, pastry, bacon, poor beer, or others, caused any great difficulty, he used the pump and washed them out—often twice daily.

"In comparison to this refined behavior of the Black Forest wool-spinner, how insignificant," says Kussmaul (l. c., page 498) "is the classic *consuetudo vomitandi* of Vitellius and other celebrated gourmands of ancient Rome, who had at command only the maneuver of the palate-tickling finger or the feather."

But, from the onset, Kussmaul was not content with the simple evacuation of the stagnating gastric contents. On the contrary he sometimes also employed the stomach-pump for irrigating the diseased gastric mucous membrane with suitable fluids, designed to have a curative effect, or to limit processes of decomposition and the proliferation of fungi (sarcina).

First, natural *alkaline waters* were employed, Vichy and Vals, which, however, were gradually replaced by corresponding artificial soda solutions. For the latter purpose, *borax solutions* (4–400), *phenyl acid solutions* (25.0 creosote water to 400 of water), solution of *sodium hyposulphite* (4 to 8 grams to 400 of water), which, it was expected, would cause the formation of disinfecting sulphurous acid in the stomach, also lign. quassiæ amar. raspat., of which 30 grams were covered with 400 grams of cold water, and, after macerating for twelve hours, the colature was employed. The action of stomach washing was assisted by *dietetic* treatment, which comprised the ingestion of small but frequent meals consisting of food which could be easily digested and contained nothing that might produce abnormal acids; also the employment of soda-containing mineral waters (Vichy, Vals) given in amounts of a ½ schoppen, one-half to one hour before breakfast or before the first meal, for the purpose of dissolving the tough mucus which had formed in surplus, to neutralize the *abnormal acid,* and to stimulate the secretion of normal gastric juice; finally, keeping the bowels open by means of sour milk, buttermilk, simple warm water enemata, and rhubarb.

The great enthusiasm aroused by the first communication of Kussmaul before the Frankfort Naturalist Congress, and, even more, the general recognition in all professional circles of the world of the wonderful success of his method of treatment, when compared with other modes, as detailed explicitly in the *Deutsches Archiv für Klinische Medicin*, led to the rapid introduction of the stomach-pump into hospital and private practice. With the publication of Kussmaul's method of treatment, voluminous literature of the subject rapidly appeared which at first brought only novelty in regard to technic, various processes being described which permitted the non-employment of the stomach-pump, as this could not always be readily obtained.

In Kiel, Dr. Schorer, the assistant of Bartels, in the absence of a Wyman pump used the ordinary laboratory air-pump. Jürgensen, at that time professor in Kiel, inserted between pump and stomach a two-necked Woulff's bottle, in order to meet the objection that all fluids discharged from the stomach passed through the metal boot, and thus corroded and damaged the pump which was very difficult to clean.

Jürgensen, however, soon noticed that the stomach-pump was only necessary until the tube was filled which led from the stomach to the low-lying Woulff's bottle: *From that time on the indications for emptying the stomach by the action of a lever were given.* Jürgensen obtained the latter just as well by abdominal pressure as by the suction pump. *For this reason the use of the stomach-pump was discontinued, the stomach was no longer pumped out but evacuated.*

In his article: "An Addition to the Local Treatment of Gastric Diseases" (in the *Deutsches Archiv für Klinische Medicin,* vii, 1870), Jürgensen describes the *simplest method* embodying the lever principle, which he advises for the busy practitioner as the most serviceable, in the following:

"By the aid of the tube which is introduced into the stomach the latter, if empty, is filled with a fluid to be utilized in washing; naturally, if the stomach contains sufficient fluid this is unnecessary; then by means of a glass tube or a hard rubber and glass tube (1.5–2 meters long, and one centimeter in diameter) an ordinary rubber tube is attached to a sound.

"*The patient now stands upon a chair, and either coughs or retches.* The flow from the stomach immediately begins. The emptiness of the stomach is indicated by a sudden jerking of the rubber tube; and the stream of outflowing water at once becomes thinner.

"Thus, in a brief time, a large amount of fluid may be forced through the stomach. To be certain that my lever may actually be filled without difficulty by the prelum abdominale, I fill the stomach with water, and pay no attention to the slight inconvenience which the patient appears to experience." (Jürgensen.)

Compared with gastric lavage as practised to-day, this method described by Jürgensen as the simplest appears to require considerable assistance from the patient who must get upon a chair with a sound in his stomach. Nevertheless, we owe it to Jürgensen that the pump was proven to be unnecessary.

Jürgensen did us the further service of attempting to substitute *soft rubber sounds* for the stiff French and English stomach sounds which were then in use.

These stomach sounds were of very small caliber, and had upon their anterior end two or four small fenestra, in consequence of which they very readily became clogged with the remains of food and even with mucus. A very disagreeable feature for the patient was that, as soon as clogging occurred, the sound was taken from the stomach, cleansed, and reintroduced. This process was improved by Jürgensen who obtained from the

factory of Galante in Paris an excellent tube having a length of 70 cm., the walls being 2 mm. in thickness, and 10 mm. in diameter. This he tied with a silk thread over an acorn-shaped ivory button 2 cm. long in such a manner that the tube nowhere extended above the level of the acorn.

In the base of the lumen of the tube the ivory button had an indentation 3 mm. deep, which was intended to receive the anterior end of a whalebone sound acting as a director to prevent its lateral deviation.

Laterally, 2–2½ cm. above the ivory button, a hole was cut in the tube corresponding to its lumen. Jürgensen preferred *one* opening to several, chiefly because of the durability of the tube. If he poured water into one opening of the sound this flowed from it in a full stream, while the sound itself, owing to the well-known phenomenon of "rebound," would move with great rapidity in the direction opposite to the outflow. By this lateral deviation of the tube, Jürgensen believed, especially by turning the sound on its longitudinal axis, that he was able to *douche* the stomach by the instreaming water.

When it was desirable to prevent the lateral motion of the sound in the stomach, a second small opening, opposite the outlet, was sufficient for this purpose.

The tube-sound was so prepared that the stylet was at first introduced into the hollow orifice of the acorn-like attachment, then the tube was drawn over it and stretched, and, finally, a clamp was attached at the end of the tube opposite the acorn, over the tube and the directing staff.

"When the sound equipped in this manner has been inserted about 5 cm. into the œsophagus, the clamp is removed, the whalebone staff (mandrin) may be withdrawn, and the soft tube is passed down into the stomach and held so that it cannot be forced up by the retching" (Jürgensen).

A further simplification of the technic of gastric lavage was the insertion, by Rosenthal, of a T-shaped tube with a cock into the apparatus, which, being placed in different positions, might be connected with the vessel containing the water, with the descending outflow tube, or the stomach-tube with the outflow tube, or the vessel containing water with the stomach-tube. A similar technic was recommended by Schiffer.

Then experiments were made with *double stomach sounds*, first by Dr. Auerbach in the Clinic at Kiel, then by Ploss, Hodgen, Apolant and others. Hodgen (St. Louis) invented a simple hard rubber pump in which the ball valves were so arranged that the mere turning of the hand in a contrary direction was sufficient to reverse the stream. Hodgen even simplified the method of emptying the stomach by connecting a rubber tube with the gastric sound, and filling it with water before introducing the sound into the stomach.

According to whether the vessel containing the end of the rubber tube is higher or lower than the stomach, fluid may be allowed to flow into the stomach or may be withdrawn from it.

Extreme simplicity seemed to be attained in the apparatus employed by Th. Biedert; it consisted of a stomach-tube, a glass tube 6 to 8 cm. long, which was inserted into the former, and which at its other extremity was connected with a long rubber tube, and, finally, with a funnel into which the water could be poured (1873). To permit the water to flow from the stomach, Jürgensen, as mentioned, still believed it necessary that the patient with the full, or even distended, stomach get up on a chair. In 1875, Holland Cotton thought it sufficient for the patient to stand during the lavage; Biedert filled and emptied the stomach by raising and lowering the funnel.

A fortunate advance in the technic of gastric lavage was the introduction of *soft stomach-tubes* by Jürgensen. These instruments had the disadvantage that their flexibility was soon lost, from the fact that a mandrin of whalebone or rattan was introduced into them to stiffen them until the larynx was passed. It is greatly to Ewald's credit to have shown (1874) in a case of nitrobenzol poisoning that even a *very soft* rubber tube, or an ordinary smooth gas tube, may be introduced into the stomach and utilized for gastric lavage. In a discussion of Oser's communication ("Die Magenspülung mittels des elastischen Schlauches," *Wiener med. Presse,* 1877, 1), Nothnagel, however, emphasizes (in Virchow-Hirsch's Jahresberichten) that he also, without any knowledge of Ewald's publication, employed in his Clinic for some years a smooth rubber tube with a funnel for simple gastric lavage.

Ewald's gas tubing was the prototype of the improved tube which later came into use,—stomach-tubes which were open below, i. e., with one central opening only. Jürgensen's ivory knob at the end of a soft tube fell into disuse when, with the technical improvement in the rubber industry, closed, rounded tubes, with one or two lateral openings, stomach-tubes patterned after the Nelaton catheter, were then manufactured. Such tubes were first employed by Leube; by and by, however, they came into general use, and have been exclusively employed in gastric lavage.

The technic of the introduction of the sound in gastric lavage which was carefully constructed and practised in Kussmaul's Clinic, had then to be learned and practised by others who desired to utilize this new method of treatment. It goes without saying that in such mostly autodidactic handling of the sound the patients must have been obliged to bear a great deal. I do not believe I am far wrong in assuming that the widely prevalent fear of the stomach-pump which still exists in the minds of many patients originated in that period when physicians were learning to use the stomach sound and the stomach-tube.

Recognizing these evils, v. Ziemssen was the first to advise care in the employment of the stomach-pump (1872). Then Biermer (1874) published a communication concerning inflammation of the esophagus and

of the peri-esophageal connective tissue following injuries from the sound. This author was later followed by Leube and others. In the introduction of the stomach sound, as well as also in the use of the stomach-pump, and in withdrawal of the sound from the stomach, now and then injuries occurred which, under some circumstances, made these procedures dangerous. It repeatedly happened that portions of the mucous membrane were torn off by the sound, sometimes fragments from 1 to 3 cm. long and 3 to 5 mm. in breadth, therefore presenting quite a surface. In none of these cases of injury to the gastric mucous membrane were serious consequences noted; now and then more or less decided hemorrhage and syncope followed, but recovery was rapid and so complete that even in those cases in which there was an opportunity later to hold a necropsy no visible cicatrices were found in the stomach. Besides these lacerations in the mucous membrane of the stomach, there are reports in literature of injuries to the pharyngeal and esopheageal mucous membranes, perforations of aortic aneurysms, hemorrhages from esophageal varices, of erosions and superficial excoriations of the gastric mucous membrane. Moreover, the mucous membrane has been perforated by using hard sounds, and the overfilling of the stomach with fluid has caused decided ruptures of continuity in the gastric mucous membrane.

The number of accidents of this kind noted in literature is exceedingly small when we consider the extensive and world-wide use of the stomach-tube. However, in the opinion of a celebrated author (Ebstein) they do not exactly represent actual conditions. For instance, the tearing away of particles of the gastric mucous membrane with a stomach sound is even now said to be much more frequent than might be assumed from the publications.

In the year 1872 Ziemssen also advised us to be careful in the use of certain sounds, particularly the black ones (French), and of such as had friable borders at their openings. Emminghaus did the same. Leube compared the action of a sharp opening in the sound with that of a hollow chisel, and he believed the central opening in the sound to be dangerous, as had previously been stated by Emminghaus. Ziemssen (1872) gave minute directions for preventing injuries with the sound as follows: 1. Measure the distance between the epigastrium and the teeth with a sound before its introduction, and mark upon the latter a line so that the instrument will not be forced so deeply into the stomach as to damage the greater curvature; 2. Never begin to pump out the stomach before previously injecting warm water (at least half a liter), unless a short time previously large amounts of fluid have been ingested; otherwise, the gastric mucous membrane may be drawn by suction into the opening of the tube, and thus be torn away.

With the general introduction into practice of the soft stomach-tube these dangers have been almost obviated. Gastric lavage may now be

regarded as a harmless procedure provided the cases are suitable for lavage and the instruments eorreetly chosen, and that we adhere to the technical rules primarily given by Kussmaul, which are still to be explicitly discussed. The greater certainty which has resulted from this manner of handling the stomach-tube no longer limits the employment of lavage to the *dilated* stomach. Kussmaul advised the employment of lavage *as early as possible* in reflex spasm, in stenosis of the pylorus, and in pathologie eonditions leading to degeneration of the gastric walls. In eonsequence of this Reich in Stuttgart (1869 and 1870) and Schliep in London treated chronic diseases of the stomach, catarrh, ulcers, and digestive disturbances from other causes with the stomach-pump, and also employed the remedies advised by Kussmaul in irrigating the affected gastric mueous membrane,—solutions of sodium bicarbonate, potassium permanganate (1 to 100, of this 2 to 4 ounces in a vessel filled with water), carbolic acid, ehloralum (an English preparation), boric acid and tineture of myrrh. In an article from Kussmaul's Clinic, " On the Treatment of Gastralgia with the Internal Stomach Douche, and Remarks Regarding the Teehnic of Sounding the Stomach," Malbranc (1878) described the anodyne effect of sprinkling the gastric wall with warm water, and particularly with warm carbonated water (100.4° F.). Rosenheim, in 1892, advised a partieular sound with many small lateral openings for douching the stomach. In the same year Löwenthal suggested in the treatment of hyperchlorhydria and gastralgia spraying the gastric mucous membrane with solutions of silver nitrate (1 to 1000) and Einhorn employed the spray in various gastrie affections.

The communications of Cahn, from Kussmaul's Clinic, upon the treatment of intestinal obstruetion by gastric lavage (1884), received merited attention. In many eases of ileus, the results of repeated, even of a single, irrigation of the stomach, as I later had abundant opportunity of convincing myself, were remarkable. The benefit was brought about by the removal of gas and fecal masses lodged above the stenosed area. This removes tension and the limitation of space. Deeided peristalsis and vomiting cease, and food may then be introduced.

I must also attribute to Kussmaul's initiative the method, which I have systematically described and praetised, of covering with bismuth the ulcerated and bleeding surfaees in the stomach, these having first been cleansed (1893).

The beneficial effect of gastric lavage upon the bowels in dilatation of the stomach, Kussmaul mentioned in his article to which we have so often referred. This was noted in all cases in which gastric lavage was at all serviceable, and he looked upon it as a suspicious sign if, after prolonged gastric lavage, amelioration took place, and the gastric symptoms improved, but constipation stubbornly persisted.

In conjoined labor with the venerable master for almost ten years, *I have availed myself of the benefits of gastric lavage in intestinal disturb-*

ance to an enormous extent, not only when constipation existed, but even more frequently when prolonged diarrheas were present which so often have their origin in disturbances of gastric function and organic disease of the stomach. Here I cannot refrain from mentioning the favorable effect of gastric lavage in many diseases of the liver, in diseases of the gall-bladder combined with jaundice, in chlorosis and anemia, in many disturbances of metabolism, and in intoxications. Gastric lavage has not only a local effect, but, in those cases where the pylorus opens during lavage, also a general effect in that the water which streams into the small intestine, if not regurgitated into the stomach, and not expelled externally, reaches the blood and the liver, washes out the blood and the vessels, and, in particular, cleanses and washes out the kidneys just as effectually as the best mineral spring cures.

Furthermore, I must call attention to the value of gastric lavage in diagnosis; this was evident from the beginning and has become still more apparent in the course of time. "We first empty the stomach, and then the palpating finger may detect *tumors* which cannot be made out when the stomach is filled. Secondly, lavage is a very valuable means by which to differentiate dilatation of the stomach from *dilatation of the transverse colon;* this was formerly very difficult, so much so that, as I know from my own experience, the greatest clinicians occasionally made gross mistakes" (Kussmaul, 1869).

Finally, gastric lavage enables us to arrive at conclusions regarding the motor condition of the stomach, and Leube did us a lasting service in utilizing gastric lavage to estimate the time necessary for gastric digestion and for the *diagnostic determination* of the motility of the stomach. By systematic washing out of the stomach a certain time after the ingestion of various foods and drinks Leube, and, following him, Penzoldt, gained absolute knowledge concerning the activity of the stomach, and the gastric digestibility of food. The results obtained became the basis of scientific dietetics. This was the origin of the well-known diet schemes of Leube, as well as the tables of Penzoldt, which are so useful in practice, showing the gastric digestibility of ordinary food and drink.

The motor function and the activity of the stomach were considered by Leube as sufficiently good if, in the course of from five to seven hours, after a test-meal at midday consisting of soup, meat and a roll, an evening washing of the stomach showed it to be almost or entirely empty. If, at this time, considerable quantities of the midday meal are still found in the stomach, digestion is slow or imperfect. Such a stomach, however, as is taught by clinical experience, will during the night rid itself of its contents and become empty by the morning even if another meal has been eaten in the evening before complete emptying. I, therefore, regard a stomach which does not become empty during the night, and in which early in the morning remains of food from the previous day are still present,

as deficient in a motor respect only. A flaccid or atonic stomach is occasionally not empty early in the morning on account of an error in diet, either by overloading and over-distending it, or because of improper food and an improper mode of life, while it may be quite capable of digesting and assimilating suitable food. On the other hand a dilated stomach in consequence of moderate stenosis of the pylorus may, for a long time—until improved by washing—show motor insufficiency, but in severe cases it is permanently in this condition, in which case it is never empty early in the morning, and is therefore incurable by gastric lavage.

At this point I shall mention what Kussmaul found to be the limitations of his method in the treatment of gastric dilatation. He was able *to cure dilatation of the stomach* by gastric lavage when no, or only moderate, constriction of the pylorus or duodenum was present. *No cure, but amelioration only,* could be brought about by gastric lavage in the following conditions:

1. In *malignant stenosis of the pylorus,*
2. In *very decided cicatricial narrowing of the pylorus,* and
3. In *moderate narrowing, when the gastric wall, in consequence of chronic gastritis, had suffered such extreme degeneration as no longer to be capable of retrogression.*

For the cases which at that time (1869) were looked upon as incurable, gastric lavage was evidently a remedy that, *if employed early,* would decidedly prolong life and markedly improve the nutrition.

"Naturally, a cicatricial narrowing which cannot be dilated even to the extent that a goose quill may pass through the pylorus, can never be cured by gastric lavage." With keen discernment, the great master continues: "*Whether in the most daring ages of a distant future, an attempt may be made to produce radical results by gastrostomy, and to form a gastric fistula and dilatation of the stricture by the knife or sound, no one can to-day positively assert.* We fear that even the proposal of such a method of relief may expose us to silent or expressed ridicule." Kussmaul, *Deutsch. Archiv für Klin. Medicin,* vi, p. 485.

Perhaps it was the inspiration of this ingenious thought of Kussmaul's which stimulated his friend Billroth (who was, unfortunately, too early removed from his sphere of activity) to attempt the cure of heretofore incurable gastric diseases, in which internal treatment had been of no avail, by operative procedures.

But this was by no means left to a later race of surgeons; by means of antisepsis and asepsis the contemporaries of Kussmaul have been enabled to operate on diseased organs within the cavities of the body. Since that time surgery of the abdominal organs—and, by no means least, gastric surgery—has had a brilliant and wonderful success. At the turn of the century the realms of internal medicine and surgery are no longer, as a few decades ago, sharply, almost diametrically, opposed to each other, but, in

united labor, such as Kussmaul saw with his mind's eye or hoped for, the physician and the surgeon are working hand in hand to reach the same high goal: To cure!

THE CLINICAL EMPLOYMENT OF GASTRIC LAVAGE

Owing to the results achieved by gastric lavage and diet, the treatment of diseases of the stomach has become one of the most grateful tasks of the internal clinician. In the interest of the practising physician it is to be greatly deplored that this realm, which is constantly extending, threatens to become the domain of specialists. For the peculiar sense of satisfaction which springs from the consciousness of having relieved the sick from distress, and which richly compensates the physician for many weary hours of his professional life, can scarcely be greater after a fortunate version or forceps extraction, after the curative removal of pus from a concealed abscess, after the removal of an obstruction to respiration by the aspiration of a pleural exudate or of ascites, or after the instrumental relief of an over-distended bladder difficult to reach—than after a gastric lavage, which frees from his painful condition an unfortunate whose stomach has no longer the power to empty itself.

The dread of disagreeable consequences which the first trial of gastric lavage brings to the physician as well as to the patient, and also the possible danger of certain unfortunate errors in technic for which we are responsible, may restrain some physician, whose position in regard to his patient is often very difficult, from employing this otherwise most useful method. Nevertheless, particularly in severe disease, the value of gastric lavage carried out at the right time is so great, and the injury which ensues from neglecting the necessary emptying of the stomach may be so considerable, that personal considerations must be set aside. Besides, with the methods in vogue to-day it is quite possible to use the stomach-tube for diagnostic and therapeutic purposes in such a way that harm to the patient may be absolutely avoided.

The technic of gastric lavage has become of such vast practical importance, for the physician as well as for the patient, that I am led minutely to detail some *old, well-tried rules* which I learned and constantly used in association with my highly honored teacher, Kussmaul.

The stomach-tube, as is well known, serves *for diagnostic and therapeutic purposes.* We test the function of the stomach to aid us in the *diagnosis* of a gastric affection or in the correct decision as to the part which the stomach performs in a complex of pathologic phenomena. This test comprises two parts: A test of the gastric contents at the height of digestion, and a test lavage.

A test of the gastric contents is made a certain time after the administration of a test-meal, usually from three to three and a half hours after

a meal consisting of gelatinous soups (250), roast beef (200), and mashed potatoes (200), or one hour after a trial breakfast consisting of tea and a roll. By means of the stomach-tube a sufficient quantity of the gastric contents is obtained for chemical, physiologic and microscopic investigation, so that we may examine the *secretory* and *fermentative* processes in the stomach.

The *trial lavage* of the stomach takes place early in the morning, the stomach of the patient being empty, and enables us to form conclusions regarding its *motor* activity, as well as also in regard to many pathologic conditions, such as catarrh, secretory irritative phenomena, continuous secretion of gastric juice, and stagnation and decomposition of food.

In the trial lavage of the stomach, which naturally should be empty, we must not neglect to secure separately the stagnating contents or the first fluid which comes from the stomach, and these should be examined chemically and microscopically—at least, with *litmus paper* and with *Congo paper*. If circumstances permit, a test of the gastric contents should first be made, and then the trial lavage should be attempted; there should be a day of rest between these procedures.

How these results assist in the diagnosis and indications for treatment will be described later.

The *therapeutic indications* obtained by the aid of the gastric tube are manifold.

Frequently the stomach-tube is used for the purpose of emptying and washing the stomach. Such stomach washings are indicated in all cases in which the stomach, early in the morning, is not empty, but in which are found remains of food from the preceding day (or even earlier), mucus or gastric juice containing hydrochloric acid such as occurs in chronic gastric catarrh, and a continuous flow of gastric juice in consequence of permanent irritative conditions of the gastric mucous membrane.

Even if the stomach early in the morning is quite empty, gastric lavage is often performed. Washing or spraying the gastric wall with the stomach-douche may be designated as the best remedy to stimulate the appetite, the flaccid gastric musculature and the secretory glandular apparatus, and also to relieve irritative conditions and pain or vomiting. All of the symptoms just mentioned are indications for the employment of the stomach-douche. Instead of water of varying temperature (usually from 86° to 95° F.) saline solution, mineral water and diluted drugs of various kinds are employed, according to circumstances.

Gastric lavage is also frequently and successfully employed in disturbances of function and in diseases of the intestine and of the liver, in conditions of abnormal blood-mixture, in renal diseases, and in acute poisoning, as well as in chronic states of intoxication, for example, uremia.

The indication for gastric lavage in ileus (intestinal obstruction) deserves special mention.

Before each introduction of the stomach-tube or of the sound the patient must be carefully examined, since this alone makes it possible to discriminate in the choice of patients, and to exclude those in whom gastric lavage might be dangerous.

In deciding what cases are suitable for gastric lavage it must be remembered that the first introduction of the stomach-tube almost invariably produces excitement, retching, and nausea, sometimes also difficulty in respiration and cardiac palpitation. Some patients, in consequence of psychic stimulation, hold the breath, become cyanotic, and, by a mighty exertion of the prelum abdominale, bring about a decided rise in blood-pressure. In irritable and anxious patients, all these symptoms appear more rapidly and reach a higher degree than in persons of more quiet temperament who are able to control themselves. The same is true of those who suffer from diseases of the respiratory and circulatory organs.

For this reason persons who, on account of diseases of the respiratory passages, the lungs or the pleura, can only complete their respiratory interchange of gases with difficulty, are markedly disturbed by gastric lavage, and, under some circumstances, to them it even becomes dangerous. The effect is similar in those who have a disease of the heart, whose myocardium is weakened or degenerated, or whose valvular disease is so defectively compensated for as to be unable to withstand great variations in blood-pressure. *Therefore, we refrain from stomach washing and also stomach evacuation to promote the diagnostic investigation of the stomach contents when severe pulmonary and cardiac affections are present.* If, however, the conditions are such as to necessitate the introduction of a gastric tube, and if it is believed that the value of this procedure will outweigh the possible danger from its use, the expert, relying upon the accurate handling of the stomach-tube while carefully regarding the rules of the technic, will, nevertheless, attempt to introduce the instrument. If the first trial is successful, with like caution others may follow, for a sensible patient soon becomes accustomed to its use. Many a phthisical patient by the washing out of the stomach has gained an appetite, acquired new strength, and prolongation of life. I even believe that *incipient phthisis* in many persons may be arrested with the removal of the anorexia by gastric lavage and subsequent plentiful feeding, and that in this way a cure may be brought about.

As a rule, we avoid the use of the stomach-tube if we have reason to suspect that the walls of the vessels in any part of the body are not able to withstand a rise in blood-pressure brought about by the introduction of the sound, which is apt to produce retching, vomiting, or decided action of the abdominal press. *Preceding hemorrhages* (hemoptysis, hematemesis, melena, apoplexy) *or a tendency to hemorrhage still present, also atheromatous arteries, valvular disease of the heart demonstrable by percussion and*

auscultation, and even suspected aneurysmal dilatation of the aorta, are valuable guides.

Recent and profuse hemorrhages from the stomach or from the lungs, high-graded atheroma, severe cardiac disease, and aortic aneurysm are always contraindications to the employment of the stomach-tube.

Capillary hemorrhages from the inflamed, perhaps also eroded, gastric mucous membrane, and parenchymatous bleeding from ulcerated, decom-

FIG. 2.—Terminal piece of a soft tube as found in the instruments usually on the market.

posing growths in the gastric wall, may, under some circumstances, also be contraindications; usually, however, they are not; but such hemorrhages generally cease if, after the discharge of accumulated and decomposed masses, the stomach is again able to contract. In hemorrhages of any kind, even during the menstrual period, I only very exceptionally make a gastric examination with the stomach-tube and perform gastric lavage. During menstruation the gastric mucous membrane of many women is somewhat inclined to hemorrhage. For this reason I interrupt the treatment by gastric lavage, and resume it only two or three days after the menstrual period. According to experience, *women during menstruation digest food less well, and they secrete a weaker gastric juice* than at other times, therefore it is well to defer the investigation of the gastric contents.

To obtain the gastric contents and for gastric lavage we employ exclusively *soft stomach-tubes,* and preferably those of English manufacture (Jaques's œsophagus tube) on account of their smooth surface and great flexibility. However, these English tubes have glaring defects which for

FIG. 3.—Terminal piece of a soft tube with a lateral opening, and a central opening in the longitudinal axis of the tube. This tube appears to me to be most dangerous to the mucous membrane of the pharynx, of the œsophagus, and of the stomach.

some unaccountable reason the manufacturers do not remedy. *Their openings are frequently not large enough, and the margins of the openings, without exception, are so sharp that they readily injure the mucous membrane, particularly the mucous membrane of the stomach,* as may be seen from Figures 2, 3, and 4, which show the configuration of the tubes in natural size. This defect may be remedied by burning out the openings, and rounding off the edges with a hot darning needle or a hot glass rod, as may be seen in Figures 5 and 6. But, after this treatment of the margins of

the openings, the rubber is rough for a time, and must be rubbed down with pumice stone. Besides, the tube has for days an odor of burnt rubber, and, sooner or later, small rents appear in the edges which have been burnt and soon render the tube unfit for use. It is to be hoped that the

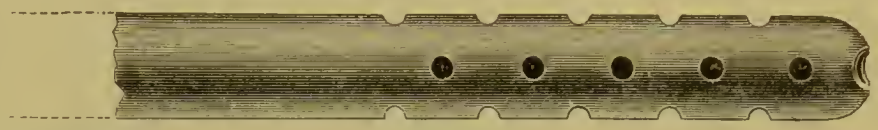
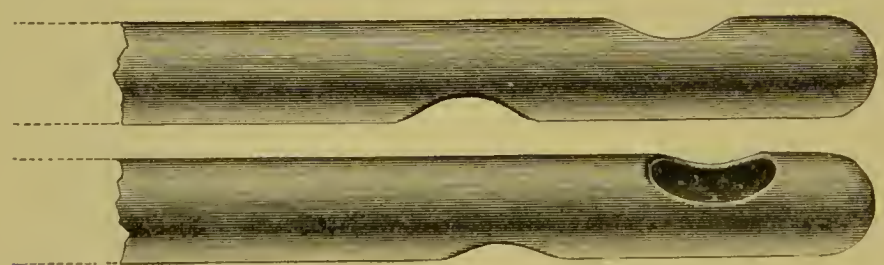


Fig. 4.—Terminal piece of a soft tube with a central and numerous lateral openings, and said to be more suitable for irrigation of the stomach than tubes with only two large lateral openings.

manufacturers will speedily place on the market more serviceable tubes with rounded, polished edges that will meet practical requirements.

The introduction of a staff or mandrin into a soft stomach-tube is decidedly objectionable, for, with a mandrin inside, the soft tube again becomes a hard sound with all of its defects and dangers. They are only required in gastric lavage and artificial nutrition when it is necessary to overcome an obstruction in the front of the stomach, in narrowing of the esophagus, or in deep-seated dilatations of the esophagus, for example, in some ante-stomachs.

Preparation of the Patient.—Before the first introduction of the stomach-tube I have never failed minutely to inform patients who are at all intelligent regarding the purpose and utility of the proceeding, to call their attention to its harmlessness, and to teach them how to conduct themselves during the operation. Whenever possible, I have always allowed



Figs. 5 and 6.—Terminal piece of soft tubes whose openings have been rounded off by a hot glass rod and thereby rendered harmless.

nervous patients an opportunity to see how willingly other patients with practice swallow the tube themselves and permit lavage.

The patient now sits erect in a chair, or, if in bed, sits upon the edge of the bed. A basin is given him in which to catch the saliva which flows from the mouth, or the vomited material, and which he holds with both hands, so as not to disturb the physician by making motions to oppose him. If dealing with patients who have very sensitive pharyngeal mucous membranes, such as smokers who retch easily and show a tendency to vomit,

the introduction should be preceded by gargling with a watery solution of potassium bromid, or by painting the pharynx with cocain, when the sensitiveness and reflex irritability of the structures of the pharynx may be so controlled that we may proceed with the introduction of the tube. Artificial teeth must, naturally, be removed. The patient should be allowed to drink some water before the lavage, so that the mucous membrane of the mouth, tongue, palate, pharynx and esophagus may be moistened and more readily permit the passage of the tube. Where there is a very irritable stomach and a great tendency to vomit, the patient should even be made to swallow a considerable quantity of water before the introduction of the tube, so that this will not reach an entirely empty stomach and, by direct contact with its walls, produce great irritation.

Naturally, if we desire to obtain the gastric contents for the purpose of analytical examination, water is not permitted before the introduction of the tube. In this case the prevention of retching and a tendency to vomit are not at all necessary for, under these circumstances, the evacuation of the gastric contents is only facilitated. In this latter operation —in case it is done without a pump or aspiration apparatus—injury to the gastric wall is unlikely, since the stomach is not empty, and the tube can hardly come in contact with the walls of the stomach.

Preparation of the Stomach-Tube.—According to Luschka's plates, the ninth spinous process of the thoracic vertebræ corresponds exactly to the position of the cardia, provided the location of the abdominal organs is normal. Before the first introduction of the tube we count on the patient's back the spinous processes of the vertebral column up to this point, to which we apply the upper opening of the tube, and measure along the vertebral column, passing alongside the ear to the incisor teeth, and mark this point upon the tube with a waxed silk thread which we tie around the tube, or with a colored pencil. If the tube is now introduced up to this point it is certainly in the stomach, provided this organ is in its normal situation, and it is unnecessary to shove the tube up and down to ascertain whether it is in its proper position, as this will cause retching and a tendency to vomit. Before the introduction is begun the end of the stomach-tube is moistened in luke-warm water.

While the patient sits erect in a comfortable position, with the head raised, the mouth open wide, and the point of the tongue placed against the lower incisor teeth, the rounded end of the stomach-tube is pushed over the dorsum of the tongue up to the posterior pharyngeal wall. The moment the tube reaches this point, the patient slowly bends his head forward and simultaneously attempts to swallow. The sound glides along the posterior wall of the pharynx, along the vertebral column to the cricoid cartilage, often even beyond this. Now we pause for a moment, ask the patient to breathe deeply and regularly, and then to swallow again, when the tube will slide downward behind the larynx, and with a slight push

on the part of the physician the instrument is rapidly introduced up to the required mark. When this is accomplished the tube is held firmly until the patient breathes quietly and regularly, and, in the meantime, a ring of hard rubber which encircles the tube, is put between his teeth, and upon this ring he may bite.

The further procedure depends upon the purpose for which the tube has been introduced.

To Test the Gastric Contents.—If it is desirable to obtain some of the gastric contents for a test of the chemism of the stomach, we direct the patient, after the tube has been introduced and is in the right position, to contract his abdominal muscles as if he were having an evacuation. By this exertion of the abdominal press, the gastric contents are brought up through the tube and may be caught in a vessel held for the purpose.

It may happen that, on pressing, only air comes up through the tube and none of the ingesta. The tube is then gently pushed further down, and after a few deep respirations renewed attempts at expression are made. With very flabby abdominal walls, the result may sometimes be brought about by pressing a hand upon the back and one upon the abdomen to assist the abdominal press, or by raising the protruding abdomen so as to bring the descending stomach and its contents nearer the vicinity of the tube.

If these manipulations are futile, either the stomach is empty or the tube is clogged by insufficiently masticated particles of meat of the test-meal which have not yet been dissolved in the stomach and are too large to pass. It is not always easy to determine which of these two possibilities is the case. If, on repeated attempts at pressing, only air comes up through the tube—and this may be recognized by the sound which accompanies it—this is in favor of the *stomach being empty.* If, on the other hand, upon attempts at pressure nothing or only the sound of air is heard, which suddenly ceases, or if the stream of stomach contents suddenly stops flowing from the tube and does not again flow when pressure is made, *the tube is clogged.* It may be that the tube is not clogged by particles of food, but that *a fold of the gastric mucous membrane is sucked into the opening of the tube* and thus closes the opening. The latter possibility must be borne in mind if, upon pressure, neither air nor stomach contents come forth.

In such cases there is danger, by rapid displacement of the tube, of injuring the gastric mucous membrane, or of tearing off a fold of the mucous membrane which has been sucked into the opening of the tube. Great care is necessary in moving or in withdrawing the tube. To be assured of the results when gastric contents are not brought up through the tube, hold it firmly, connect it with the other parts of the lavage apparatus, and permit a measured quantity of lukewarm water to flow into the stomach. If this water, upon lowering the funnel, regurgitates quite

pure, the stomach has passed the test-meal into the intestine, and the tube may now be removed with the observance of the necessary preeautions. The attempt to obtain the gastric eontents has, under such circumstances, failed, and if we desire to obtain further insight into the ehemism of the stomaeh the test-meal must be repeated at some other time, and at an earlier hour. At all events, the operation gives proof of a nor-mal, even increased, motor activ-ity (hypermotility) which com-pensates for secretory disturb-anees.

A proof that the tube has been clogged, and that the stom-ach is not empty, is the fact that water poured into the stomach reappears mingled with gastric contents when the funnel is low-ered. Qualitative investigation for hydrochloric acid, at least with Congo paper, may be made in the fluid thus obtained, but no *quantitative results* are fur-nished. Under these circum-stances, therefore, another test-meal must be given, the attempt be repeated another day, with due care that the meat has been fine-ly ehopped so as to avoid clog-ging of the stomach-tube. In the first attempt, I do not have the meat for the test-meal chopped, for it often aids us in the etiology and treatment of many digestive disturbanees to know how the patient ehews.

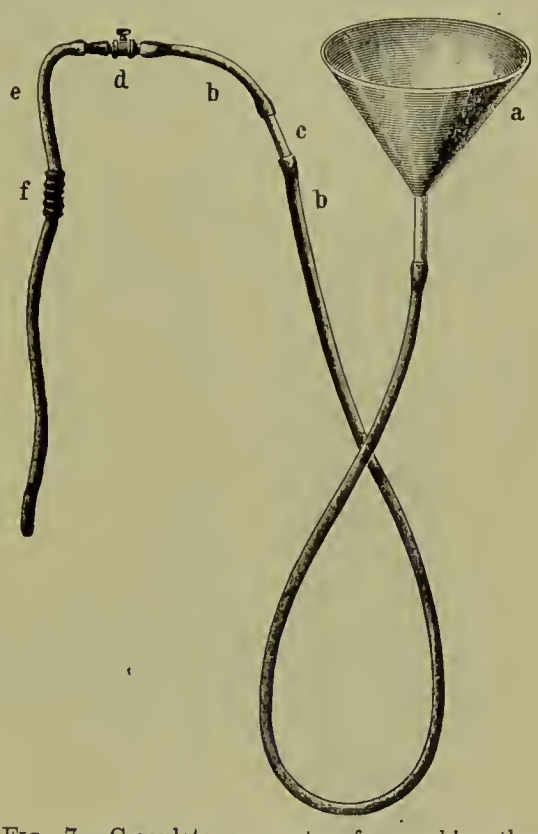


Fig. 7.—Complete apparatus for washing the stomach. (After Kussmaul.) *a*, funnel; *b*, rubber tube, one and one-half meters long; *c*, glass connecting piece; *d*, enclosed piece of hard rubber with cock; *e*, stomach-tube; *f*, ring of hard rubber on which to bite.

Gastric Lavage.—For stomaeh washing we require an apparatus (Fig. 7) which is eomposed of the already mentioned stomaeh-tube and the ring which is placed over it and held between the ineisor teeth (Fig. 8) for biting, a glass funnel holding from one-half to three-quarters of a liter, and a rubber tube one and one-half meters long interrupted in its lower third by a short glass tube which is eonneeted with the funnel, also an inserted piece by which the rubber tube is readily attached to the stomaeh-tube. This enclosed piece (Fig. 9) is made of hard rubber, and is of the form and size depieted in the illustration. The round end, *a*, is placed toward the funnel and attached to the rubber tube. At *b* there is a faucet which is usually firm and air-tight, but, under eertain eonditions which we

will learn to recognize, may be removed for a short time. The conically narrowing end, *c*, of the inserted piece answers the purpose of connecting the tube with the stomach-tube and, on account of its shape, fits a tube of any caliber.

If the stomach-tube has been introduced according to the directions given, the biting ring is between the incisor teeth, and everything else in position, we fill the lowered funnel with lukewarm water, and then raise it so that the water flows into the stomach. When proper assistance is given I permit the funnel to be filled as soon as the tube has passed the larynx and before it is introduced and elevated.

Fig. 8.—Ring on which to bite (of hard rubber) drawn over the sound, and readily moved about upon it.

The rapidity with which the water flows into the stomach depends upon the pressure of the column of water, i. e., upon the elevation of the funnel, upon the degree of contraction of the gastric musculature, and upon the pressure of the prelum abdominale. Under some circumstances, as in retching or coughing, nothing flows into the stomach, and even when the funnel is raised very high either the water for washing or some of the gastric contents returns through the tube. On the other hand, if the stomach walls are flaccid, on deep inspiration and in consequence of the diminished intra-abdominal pressure the water is aspirated into the stomach and actually streams into this organ. It may then occur, if the funnel is raised high, that a bubble arises and by means of this, simultaneously with the water, air is aspirated into the stomach and causes an over-distention of the organ. A simple artifice, namely, *slanting the funnel,* may prevent this difficulty.

Before the funnel becomes quite empty, it must be rapidly lowered so that no air can pass into the tube, and the continuous flow of water through the tube is not interrupted. On lowering the funnel, a siphon action takes place, and the water regurgitates into the funnel. It may now be collected, examined, its reaction tested with litmus or Congo paper, and we should carefully observe whether as much water flows out of the stomach as has been poured in. By this precaution, it is quite impossible to damage the stomach by over-filling it with fluid. In the stomach-tube and in the wash-tube, even after emptying the funnel, a column of fluid remains. A glance at the glass tube within the stomach-tube will show of what this fluid column consists. If it is clear, or but slightly turbid, we should continue the lavage. The lowered funnel is refilled with water, is raised and lowered again, the water used for washing is collected and emptied, as before.

If, however, the fluid in the tube is quite turbid, or decidedly mixed with mucus, remains of food or the like, the faucet at the inserted piece is removed for a moment so that air enters the tube and the contents flow toward the funnel. When this is done, the faucet is reintroduced, the washing is continued, the faucet is again removed, and if the water returned a second or third time through the funnel is not yet clear, this process is continued until the water flows clear. Without the enclosed piece with the faucet—which, by the way, is the invention of an artillery officer who was treated in Kussmaul's Clinic for dilatation of the stomach—the soiled water is again forced through the tube and returns to the stomach. The process is then unnecessarily prolonged, and this is an evil of some consequence in debilitated patients, one which is obviated by the introduction of the enclosed piece, which is as simple as it is ingenious.

The stomach, as a rule, is washed until it is clean, provided the patient is able to bear it. In very irritable patients we must reckon upon their resistance, and, particularly in the first lavage, we may be obliged to desist

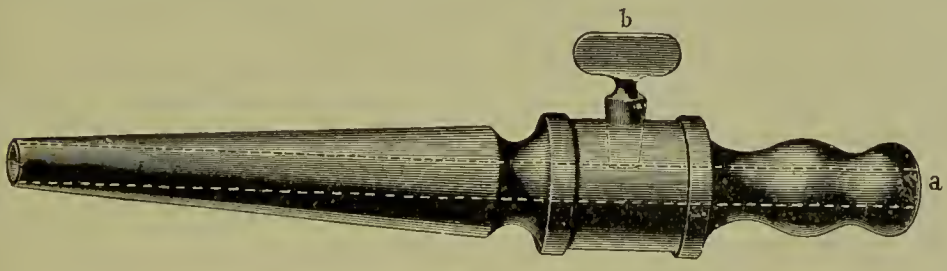


FIG. 9.—Inserted piece of hard rubber with cock.

before this end is attained. Even with patients whose activity and self-control is only slightly diminished, days frequently pass before the remains of food from former meals can be removed from a dilated stomach.

Besides, a return flow of clear water does not always indicate that the stomach is actually clean. For example, *mucus,* which often thickly coats the gastric walls, *dissolves but slightly or not at all in pure water;* the water may, therefore, flow off clear while a mucus coat, rich in microbes and ferments, may still adhere to the gastric mucous membrane. If, in such cases, alkaline waters or solutions of soda and table salt are used for washing, it is frequently astonishing to see the amounts of mucus brought up.

The conditions are somewhat different in a flaccid and dilated or abnormally-formed stomach. When the patient is in a *sitting posture,* the fluid reaches only the *lower segment of the flaccid sac,* and sprays the posterior gastric wall and the small curvature, also the region of the cardia and the fundus, slightly or not at all. If the patients with gastric dilatation who have become accustomed to the sound are made to lie down after an apparently satisfactory washing carried out in a sitting posture, and

lavage is continued while in the recumbent posture, surprisingly large quantities of mucus and remains of food are brought up with the water. Those who have become accustomed to lavage in the recumbent posture bear it very well, so that, after the stomach is filled with water, this fluid may be moved to and fro in the stomach by movement of the trunk. The gastric wall is thus thoroughly washed up to the small curvature, which is particularly important since this area is the preferable seat of the severest gastric diseases.

In hour-glass stomachs, those with deep diverticulum-like sacs formed by cicatrices, also in such as have a markedly developed antrum pyloricum and antrum cardiacum, we sometimes observe that, after an apparently thorough gastric lavage, turbid gastric contents which have been enclosed in a sac suddenly pour into the funnel. The symptom may frequently be utilized in diagnosis.

In removing the stomach-tube when the washing is finished, we will note that, with a lowered funnel, a column of fluid remains in the tube because the gastric wall contracts firmly around the lower end of the tube and closes the opening. By the siphon action, folds of mucous membrane may even be drawn into the opening of the tube, and, should this occur, the flow of water will cease suddenly with a jerk which may often be felt throughout the entire tube. If, at this moment, the stomach-tube should be rapidly withdrawn, there is great danger of *injuring the mucous membrane,* or even of *tearing off a portion of it that has been sucked into the tube.*

This danger may very readily be averted by holding the tube without moving it, pouring a little water into the funnel which is raised, and then slowly withdrawing the tube, when water will again flow into the stomach, and the mucous membrane be forced away from the opening of the tube. When the tube is so far withdrawn that the opening is above the cardia we compress it in order to prevent any fluid from entering the air passages while the tube is passing over the epiglottis.

Immediately after lavage the patients should have some breakfast and then rest for a little while; they should make no exertion whatever for at least an hour afterward.

Individualization and Interruption of Gastric Lavage.—Individuality of character and will power exert quite an influence upon the course of operations, which, like most stomach washings, necessitate some assistance on the part of the patient. Where this influence is a deleterious one, the physician, by his personality and skill, may be able to overcome it after a time.

Moreover, there are definite pathological conditions to be considered in the case, the influence of which upon the course of the operation cannot be foreseen nor always prevented, and, under some circumstances, they may be so great that lavage must be discontinued. We refer particularly to

the danger of extraordinary irritability of the gastric mucous membrane and the danger of hemorrhage.

According to experience lavage is best borne by patients with gastric dilatation, therefore by those to whom this operation is most necessary. The mucous membranes of the stomach and esophagus are so benumbed by the intense irritation of the stagnating gastric contents and by vomiting that they do not react to the irritation of the tube and the cleansing fluid. Hence the danger of injury to the mucous membrane by the stomach-tube in gastric dilatation is very slight.

The condition is different in irritable stomachs which are not dilated, which are sometimes even decreased in size (contracted or concentrically hypertrophied), which react by a decided, sudden contraction, by retching and vomiting, or by cough, to the least irritation; for example, to the slightest movement of the introduced stomach-tube, to a touch of the latter against the stomach wall, to the inflow of the fluid, or to the temperature or composition of the same. Vomiting in such cases often has a peculiar expulsive character. The water suddenly gushes from the mouth with great force so that care is necessary in handling the stomach-tube.

In such irritable stomachs another grave danger is that, after a sudden attack of retching with severe vomiting, or after an attack of coughing, the gastric wall is forced against the stomach-tube, and consequently *the mucous membrane is rubbed against the opening of the tube and becomes excoriated,* or *a fold of the mucous membrane is sucked into the opening and torn off,* or, if the stomach-tube has not been adjusted carefully enough, it is forced out of the stomach.

These stormy reactions of extremely irritable stomachs frequently cease if the patient obeys the command to breathe deeply and regularly. In other cases, the stream of luke-warm water flowing from the funnel held as high as possible quiets the irritable gastric walls. If this is not the case, it is risky to continue the lavage. It is wise to observe all precautions; we, therefore, slowly withdraw the stomach-tube, and defer the operation until another morning. Before attempting it again, we should never neglect to call the attention of the patient to the mistakes of the last washing, to caution him to breathe deeply and regularly, and to avoid this or that bad habit. If catarrh of the pharynx is present, this also should receive the necessary treatment.

In another category of gastric affections, lavage is disagreeable, and even dangerous, because of the *great tendency of the gastric mucous membrane to hemorrhage.* In the accidents during lavage which have been described above, hemorrhages may also occur, but only in consequence of previous gross mechanical injuries. Here we are dealing with *hemorrhages which occur without such injuries.*

There are certain catarrhs of the stomach in which the mucous membrane, particularly at the height of its folds, is enormously hyperemic.

The dilated capillaries of the mucous membrane mostly show such an extreme permeability that quite insignificant variations in pressure are sufficient to cause mucous membrane hemorrhages. Among many thousand cases of lavage, I have known about ten such hemorrhages to occur. Although they were slight they necessitated the interruption of the operation and, because they recurred with every attempt, I was compelled to discontinue lavage entirely in these cases.

In hysterical patients vomiting of bloody masses and of pure blood occasionally occurs without any loss of substance of the mucous membrane. Therefore, in such patients also, we may be forced to withdraw the stomach-tube on account of the hemorrhagic staining of the water; the same is true of gastric crises of spinal origin.

Hemorrhages from esophageal varices must also be referred to. They are more profuse than the previously mentioned form, and closely resemble the hemorrhages originating from gastric ulcers. In a noteworthy case of this kind which was at first believed to be ulcer of the stomach, in which, however, this diagnosis was abandoned on account of the absence of all other symptoms of ulcer, the hemorrhages ceased entirely under the influence of lavage. Here, apparently, the introduction of the tube compressed the ectatic veins of the esophagus. Similar conditions are seen in hemorrhage from the ureter, where catheterization or sounding acts as a potent curative factor.

If, during lavage, *congealed blood is found in the stomach* such as is observed in ulcerating carcinoma, in old ulcers of the stomach, and, particularly, where there is a stenosis of the pylorus with a spastic closure of the pylorus caused reflexly, producing hypersecretion, there is *no reason for interrupting the operation.* On the contrary, in cases of this kind, the thorough emptying and cleansing of the stomach is an exceedingly beneficial measure, and well calculated to check an old hemorrhage and prevent a new one. The empty and clean stomach is again able to contract properly, and by contraction the bleeding surface becomes smaller, the borders of the ulcer are approximated, and the gaping lumina of the vessels closed. The fresh, and often not inconsiderable, losses of substance caused by the tearing off of particles of mucous membrane by the tube most distinctly show the mighty styptic influence of the muscular contraction of the stomach. In the cases that have been reported, the hemorrhage has ceased rapidly and no ulcer formation has taken place apparently for the reason that the margins of the wound, in consequence of muscular contraction, have approximated at once, have closed and healed, like operative wounds of the stomach, by first intention.

If, therefore, during lavage, a *fresh hemorrhage* should unexpectedly appear and be indicated by the red discoloration of the water, the physician must have the presence of mind not to withdraw the sound at once; he must very gently, even though with some force, allow the stomach

to empty itself, and thus make it possible for it to contract completely. Naturally the patient in whom this has happened must be treated for a time after his hemorrhage as in the case of ulcer.

The remedies used in lavage vary with the requirements of the individual case.

For simple cleansing of the stomach, lukewarm water is sufficient (at about the temperature of 86° F.); if, however, we wish to act upon the diseased gastric wall, additions must be made to the fluid, above all, soda for gastric catarrh, and table salt for deficient hydrochloric acid. A mixture of these salts is most serviceable:

℞ Sodium carbonate..... part 1
Sodium chlorid..... " 2
M. D. S.: To be dissolved in water (2 to 5 per 1000).

But even water alone may act in different ways upon the gastric wall; lower temperatures (less than 86° F.) stimulate and irritate; higher than 86° F., lessen irritation, have a soothing effect and relax. Under the influence of warm washings the pylorus opens, even when spastically closed, more quickly than under the influence of cool washings. If in the individual case, lavage of the stomach requires a long time, it is better for the patient not to have the water too cool, as thus too much heat may be withdrawn from him. Debilitated persons for this reason bear warm washings better than cool ones.

The pressure exerted during lavage has no inconsiderable influence upon the diseased gastric wall. In all the diseases in which lavage must be carried out cautiously, therefore in gastric ulcer and in corrosion of the stomach after poisoning with a tendency to hemorrhage and extreme irritability, the funnel must not be held too high,—not above the height of the patient's head. When we wish to produce a stimulating effect by the gastric washing (gastric douche), it should be held as high as possible.

After cleansing the stomach, *bitter tonics* are sometimes sprayed upon the mucous membrane, and are allowed to remain in the stomach in contact with the gastric mucous membrane for a few minutes; among these *quassia amara* (30 grams in half a liter of cold water, macerated overnight, and filtered early in the morning), *hop tea* and infusion of dried hops, and condurango are most recommended. I employ one to two teaspoonfuls of the fluid extract of condurango to half a liter of lukewarm water.

Abnormal fermentative decomposition in the stomach is best combated by thorough and regular lavage. We possess no reliable remedy for the relief of abnormal decomposition, fermentation and other processes of decomposition, which, without danger to the organism, can aseptically change the digestive processes in the stomach and intestine. In an abnormal production of organic acids in the stomach I have most often employed washings with a 1–1000 salicylic acid solution.

Where there is an immoderate exeretion of hydroehlorie aeid (hyperchlorhydria), and in gastrie uleer, some authorities reeommend spraying the stomaeh with a 1–1000 silver nitrate solution. In severe, old, gastric uleers particularly, excellent results have been obtained, after all internal remedies had failed, by pouring bismuth into the previously eleansed stomaeh. With an approximately exact diagnosis of the seat of the ulcer, or after a few attempts with the patient in a suitable posture, it is possible to introduce into the adult stomaeh, by sedimentation, a mixure of bismuth in suspension (10 to 20 bismuth subnitrate to 200 of water) so that a preeipitate of bismuth eompletely eovers the uleerated surfaee. That a eoating of bismuth remains upon the rough or sinuous ulcerated surfaee, and irritation by the gastrie eontents is thus preventeđ, I have eoneluded from numerous clinical observations; for, after a few applieations of bismuth in the eases in question not only the sensory but also the secretory and motor irritative phenomena (pains, hyperehlorhydria and hypersecretion, peristaltie unrest and spasm of the pylorus) were lessened, and often entirely eeased, and with the eontinuanee of the bismuth treatment of the gastrie uleer for several weeks eure finally took plaee. In fresh gastric uleers and in hemorrhage, whieh do not permit lavage, I have had good results by letting the patient drink bismuth in suspension after the stomaeh had been cleansed in a natural manner by the drinking of an alkaline water half an hour previously. (Here the remark may be permissible that mineral water eures, in faet, the drinking of natural mineral waters, may be designated as natural gastrie lavage in eontrast to the artifieial, that is, gastrie lavage performed with the stomach-tube. For obvious reasons the latter is mueh more effeetive than the former.)

The *best time for gastric lavage* may be seen from the indication: *If the stomach early in the morning is not empty, it must be washed out.* At this time the stomaeh washing is least exhaustive, is eompleted most rapidly, and is most useful to the organism. The cleansed stomach-wall, refreshed by the washing with water, is ready for new and thorough activity (similar to the mouth after brushing the teeth).

It is true it is very diffieult for the busy praetitioner so to divide his time that his gastrie patients may have regular treatment early in the morning and at the same hour daily. Yet it is not right that lavage should be performed at any time during the day when our visiting list brings us to the patient. If, for example, we wash out the stomaeh at a time when the labor of digestion is not yet completed, added to the abrupt interruption of the digestive processes is the disadvantage that, besides the digestive juiees, larger and smaller quantities of unused chyme are washed out of the stomaeh.

Besides, gastrie lavage at an improper time is much more exhausting than when the stomaeh is empty. The expulsion of the insufficiently split-up food is very diffieult, the washing, therefore, takes a long time, large

quantities of fluid are neeessary to eleanse the stomaeh, mueh heat is thereby withdrawn from the patients and they are oftcn exhaustcd before the stomach is clean; *organ and organism therefore suffer injury by lavage at an unsuitable time.* The stomach becomes worse instead of better, the general condition of nutrition falls instead of rises; in spitc of patienee and labor the treatment is unsueeessful, and the eritieism is made, " Even lavage did not help him."

Moreover, the ordering of a test-meal and the later removal of the gastric contents should not occur at any arbitrary time. The stomach is not a retort into which, at any time, something may be put, or from which something may be taken to see what process it has undergone in the meantime.

I do not deny that in the treatment of diseases of the stomach our adherence to the most useful methods and to a definite time is a hindrance to the general employment of gastric lavage, and to a certain extent limits its utility in practice. It is due to these circumstances, and not to the knowledge of the practising physician, that the results of local treatment of the stomach in well conducted institutions are usually better than in private practice. Where it is at all possible, therefore, if the stomach-tube is to be employed for a long time, the patient should at onee be taught to use it for himself.

Besides, the essential point in treatment of the stomaeh is not alone the correet use of the stomaeh-tube: *Much more difficult than this* is the *art of nourishing the patient. If nothing injurious is introduced into the stomach, less will be brought up by lavage; by a proper diet, therefore, we may limit lavage and in many cases make it unnecessary.* Naturally, dietetic treatment in institutions in whieh food is prepared under the direction of the physician is easier than in private practice.

Only definite symptoms should cause us to deviate from the general rule of washing out the diseased stomach early in the morning when empty. There are cases of meehanical insuffieiency of the stomaeh in which secretion of hydrochloric acid (hyperseeretion), the formation of organic acids, and, under some circumstances, the production of gas is so great and the difficulties arising from these conditions so decided that by a rapid emptying of the stomach not only are the sufferings of the patients relieved but danger is averted.

If, in cases of this kind, examination by the physieian at any hour of the day makes it appear that the stomach should be emptied immediately, it is best to choose for lavage a late evening hour; by lavage at nine or ten o'clock, or even later in the evening according to the time when food was last taken, the patient is relieved for the night from his discomfort, and secures a very necessary and grateful night's rest. Evening lavage, however, if continued for a long time, is not well borne, not nearly so well as that of the morning, and in cases in which *both morning and evening*

lavage are necessary the patient soon loses strength, the intestine no longer receives sufficient fluid and nourishment from the stomach, the requirements of the economy of the body can no longer be met, even although we use supplementary rectal alimentation, and unless an operation is performed in such cases the patients slowly succumb to exhaustion.

FUNCTIONAL DISEASES OF THE STOMACH

By H. LEO, Bonn.

The stomach differs from other organs in that it frequently presents subjective and also objective symptoms of disease which are not due to any organic change, or, at least, not to any which are recognizable by our present methods of investigation. In this respect, only the heart, that is, the circulatory apparatus, and the intestines show similar conditions. These clinical pictures may be grouped under the term, *functional diseases of the stomach.*

One of these groups presents well-developed anomalies of the gastric contents which are not, however, due to disease of the stomach wall, but to the ingestion of tainted food, or to the invasion of microbes which in themselves cause the decomposition of the ingesta in the stomach. These morbid conditions have been designated *dyspepsia.*

The second group is distinguished by clinical pictures which present either subjective symptoms dependent only upon alterations of the stomach or, combined with this, disturbances of gastric activity which may be objectively recognized. The majority unquestionably depend upon abnormal functions of the nervous apparatus, and these affections are therefore designated *neuroses of the stomach,* in spite of the fact that a direct relation to diseases of the nervous system does not always exist.

The differential diagnosis of individual diseases belonging to this group from one another and from other organic diseases of the stomach is not always easy. To accomplish this an investigation of the entire organism is, as a rule, necessary, and of the various disturbances which uncommonly often disturb the gastric activity, or may be due to abnormalities of the latter. Naturally the chief stress is to be laid upon the special examination of the stomach, in an anatomical as well as functional respect. On account of the increased importance of this in the last few years, it appears advisable to discuss somewhat minutely the best method of examination.

TO TEST THE FUNCTION OF THE STOMACH

Of the three functions of the stomach, the motor, the secretory and the absorbent, *the motor,* i. e., the intimate admixture and mechanical preparation of the food and its timely removal through the pylorus into the intestine is unquestionably the most important. The disturbances of this

function, manifested by an increased or, usually, by a decreased action, and which appear in the most varied diseases, are then conspicuous to a high degree. To differentiate them, besides other complicated and less definite methods, it is sufficient to prove by the Leube-Riegel test-meal (see below) or by the Ewald-Boas test-breakfast (see below) that the duration of digestion deviates from the normal. As a decrease in the motor function (*insufficiency of the stomach*) primarily occurs in gastric dilatation, I shall forego a full explanation of the test for this, and instead refer the reader to the article by Riegel. It is sufficient to state here that six hours after a test-meal, and, at the latest, two hours after a trial-breakfast, the stomach normally propels its contents into the intestine. If, therefore, at this time, by sounding or by lavage ingesta are still found, the motility of the stomach is diminished. On the other hand, experience teaches us that the stomach, under normal circumstances, three hours after a test-meal and one hour after a test-breakfast, still contains remains of food. If, at this time, it is already empty, we have proof of an increase of its motility.

While a test of the absorbent function of the stomach has, up to the present time, produced no results which are of value in the diagnosis of gastric disease, the study of the *secretory function* by an examination of the constituents of the gastric secretion or of the admixture of the same with the intaken ingesta, and also of the normal and abnormal products of decomposition, is of great practical importance. Only since the examination of the gastric contents was incorporated into the diagnosis of gastric diseases have certain well-characterized clinical pictures (hypersecretion, hyperacidity, achylia) become known. Their results in many cases determine the diagnosis, either because we find changes in the gastric contents which are characteristic of certain diseases, or because changes of this kind are absent; that is, normal conditions exist, and we are thereby justified in excluding some diseases which come into question in the differential diagnosis.

In regard to the *indications for the examination of the gastric contents*, this procedure, that is, lavage of the stomach, aside from the contraindications to be at once named, is absolutely harmless and, as a rule, may be carried on without difficulty, but is occasionally unpleasant to very sensitive patients, and, naturally, not in place when other diagnostic aids permit us to make a positive diagnosis. The artificial removal of the gastric contents is now only performed with a soft rubber tube, but is absolutely contraindicated if hemorrhages have occurred even although a long time have elapsed since the last one. Otherwise, a fresh hemorrhage might readily be produced.

The stomach contents are withdrawn either when the stomach is empty, therefore, as a rule, early in the morning, or during the period of digestion; if we suspect hypersecretion (see below) it is done at the time first named. In these cases, we find a considerable amount of clear fluid while

the stomach normally should be empty. Only when ingesta reach the stomach, that is, when present there, both in the healthy and in the sick, do we find besides hypersecretion a secretion of the gastric juice. In the examination of the empty stomach the proof of gastritis may also be valuable on account of the mucus present in the opening of the tube, and which contains profuse amounts of leukocytes and epithelium. This is also the case with the proof of insufficiency of the stomach, in which the stomach that should normally be empty in the morning still contains ingesta. In all other cases, we examine the gastric contents during the time of digestion.

During this process two objects must be borne in mind: First, the determination of abnormal processes of decomposition in the gastric contents, and secondly, the determination of the properties of the gastric secretion.

To determine the first, that is, the presence of abnormal products of decomposition or causes of decomposition, it is often sufficient if we obtain the gastric contents for examination upon the first visit to the patient, immaterial when and what the patient has last eaten. But it is advisable to do this only when we desire qualitative proof of the composition of the gastric secretion (hydrochloric acid and pepsin).

This, however, is unwise when an exact estimation of the activity of the gastric parenchyma is to be made, particularly if we wish to determine the proportion of the constituents of the secretion, and, especially, of hydrochloric acid. The composition of the gastric contents, which represent a mixture of the gastric secretion and the food that has been consumed, is dependent upon the amount and nature of the ingested food and upon the time which has elapsed since the food was eaten. Therefore, if we desire to ascertain the deviations of this composition from the normal, these deviations not being dependent upon accidental variations in the intake of food, but upon anomalies of secretion, we must be sure, in the first place, that the composition and amount of the introduced ingesta are always alike, and that the stomach contents are always obtained after the same lapse of time following the ingestion of food.

For this purpose it is, above all, necessary that the stomach be empty before the foods which are to cause a secretion of gastric juice are eaten. The best time for the so-called test-meal is, therefore, early in the morning, or in the course of the morning, several hours after breakfast has been taken. When it is doubtful whether the stomach is empty it must be washed out before the test-meal is given. Of the many test-meals which have been advised to stimulate gastric secretion, I shall here mention only the two which are in most common use, which are the best, and quite sufficient for all practical purposes.

Test-meal according to Leube-Riegel.—Toward midday the patient partakes of a plate of beef-soup, 150 to 200 grams of beefsteak, 50 grams of mashed potato and a roll. Four hours later, as a rule, the stomach is empty, but it is occasionally advisable to empty the stomach somewhat

sooner or later. This method has the advantage that the gastric mucous membrane is stimulated in a normal manner, because all necessary foods are contained in the meal. It should, therefore, always be employed when the simpler *Ewald method* gives a negative result in regard to hydrochloric acid and pepsin. In such cases we often note that the more intense and prolonged stimulation of Leube's test-meal gives a conspicuously positive result; its disadvantage lies in the fact that, because of the complexity of the components of the meal, complete uniformity of the same in every case can only be obtained with difficulty, and gastric patients in particular are frequently unable to consume it entirely. In office practice it is most unpleasant, but sometimes necessary, to evacuate the stomach a long time after the meal, at a late afternoon hour.

Test-meal according to Ewald and Boas.—Early in the morning, or at another time when the stomach is empty, the patient receives one or two rolls of wheat bread (35 to 70 grams) and one or two cups of tea without sugar or milk. Water may be taken instead of tea. After an hour or an hour and a quarter (according to whether the single or double quantity has been taken) the stomach is evacuated. This test-meal has the advantage of great simplicity, and the masses which are evacuated do not have the smeary composition which they have by Leube's method. As the evacuation may occur comparatively soon after the intake of food, the administration of the trial breakfast and the evacuation of the stomach contents may be undertaken during the office hour.

[The shredded wheat biscuit makes an excellent test-meal. It is free from yeast and contains the entire food elements of wheat. Two biscuits, thoroughly masticated, and a glass or two of water form an ideal Ewald meal.—Ed.]

EXAMINATION OF THE GASTRIC CONTENTS

A. MACROSCOPIC AND MICROSCOPIC EXAMINATION

The *odor* of the gastric contents normally and also in most pathologic cases, provided the stomach is empty before the test-meal is administered, is not distinctive. In stagnation of the ingesta (gastrectasis, dyspepsia, gastritis) we find, as an expression of decomposition, the odor of fatty acids, acetic acid or butyric acid (like rancid butter).

Blood is occasionally admixed with the stomach contents as a small, bright red streak; it originates from retching, and is therefore of no pathognomonic importance. If, however, the entire contents have a distinct hemorrhagic discoloration, the presence of an ulcer, carcinoma, or stasis in the portal vein system is proven, and all further sounding is to be avoided.

Bile, characterized by its yellow or greenish color, is very frequently found in the gastric contents and is of no diagnostic significance.

Mucus in small amounts may also be found in the normal gastric contents if the quantity of hydrochloric acid present is slight. Larger amounts, particularly thick coagula, denote gastritis, unless the mucus originates from the respiratory passages. In the case of gastritis, the microscopic examination of the mucus as well as of the

stomach contents evacuated from the empty stomach is of importance. The microscope reveals numerous well retained leukocytes and epithelia; in acid gastritis in which the protoplasm of the leukocyte is digested, numerous cell nuclei are found while these normally are present in only slight numbers (A. Schmidt).

The *macroscopic composition of the ingesta* is of special significance. If, after Leube's test-meal, the particles of meat are split up, the production of hydrochloric acid is sufficient, perhaps even increased. On the contrary, if they appear unchanged there is a deficiency of hydrochloric acid, perhaps of acidity. Insufficient maceration of the fragments of bread favors hyperacidity as a greater amount of HCl inhibits the saccharifying action of the diastatic salivary ferment which normally occurs in the stomach.

The *microscopic examination of the gastric contents* may be of diagnostic weight on account of the previously mentioned formed elements in gastritis. The presence of large numbers of microörganisms denotes stagnation of the ingesta, but it must be remembered that the yeast cells may be due to the ingested bread.

B. CHEMICAL EXAMINATION

The gastric contents to be examined must be undiluted; except in an examination for ferments, it is unnecessary to filter them.

The examination primarily is for the acids, particularly hydrochloric acid, volatile fatty acids, and lactic acids. In most cases this is sufficient. Under some circumstances (if achylia be suspected), an examination of the ferment, perhaps also of the products of digestion, is expedient.

ACIDS

(a) *Qualitative Tests*

Reaction.—The evacuated contents of the empty stomach may normally have a neutral or even an alkaline reaction (the presence of bile and intestinal juices in the stomach). If, after a test-meal, blue litmus paper is not reddened, the condition is one of insufficient or absent hydrochlorie acid secretion.

Hydrochlorie Acid.—The test is first made with the *Günzburg reagent* (two grams of phloroglucin, one gram of vanillin, thirty grams of alcohol); a few drops of this with an equal quantity of gastric contents are placed in a porcelain dish and heated over a small flame. If a beautiful red margin appears, the presence of hydrochloric acid is proven. If, however, the red color does not appear, the absence of hydrochloric acid is by no means certain, as its presence, even in decided amounts, may be obscured by other combinations.

This error cannot occur in *the test with* $CaCo_3$; therefore, this test must always be used if the reaction with the Günzburg test is negative. For this purpose, we mix in a watch-glass some of the gastric contents with a pinch of powdered $CaCo_3$. The reaction of the mixture is tested with blue litmus paper, and this is compared with the original reaction of the gastric contents. If the redness of the litmus paper after treatment with $CaCo_3$ is less intense than before, or if no reaction occurs, the stomach contents (in the presence of volatile fatty acids and of lactic acid) contain hydrochloric acid. If the reddening is the same, before and after, no hydrochloric acid is present.

For further confirmation of this test *the digestion test* may be employed (see below).

Volatile Fatty Acids.—Several c.c. of the gastric contents are heated in a test-tube to boiling, while a piece of blue litmus paper is held over the test-tube. If

the paper redden, the presence of volatile fatty acids is proven: if no redness appear, they are absent.

Lactic Acid.—About 10 c.c. of gastric contents are shaken up with about the same quantity of ether, and the ether which has taken up the greater part of the lactic acid present is poured off. To the ethereal fluid a few c.c. of a very dilute ferric chlorid solution are added (one drop of an officinal ferric chlorid solution to a test-tube full of water). Even if only minute quantities of lactic acid are present, upon shaking a distinct yellow color appears in the ferric chlorid solution.

(b) *Quantitative Estimation*

We must always remember that, in spite of many methods for this estimation, we do not determine the absolute amount of acids excreted in a certain time by the quantitative estimation of the acids, but we measure relatively the secretory function of the stomach, for the reason that we never know how much of the gastric contents has entered the intestine during the time in which we are evacuating the contents, and this, therefore, cannot be calculated. As, normally, there is considerable variation in the secretion of hydrochloric acid in the same and in different persons, we should only regard marked and constant deviations as pathologic.

If the presence of HCl and the absence of volatile fatty acids and lactic acids have been proven in the manner described above (which is the case in the majority of tests), this is sufficient to determine the total acidity for practical purposes. On the other hand, if we desire to learn the exact proportion of acids present, their estimation must be included. Upon the whole, although the qualitative proof of organic acids in fermentative processes is so important, their quantitative estimation is of no great practical utility.

Total Acidity.—To a definite quantity (5 to 10 c.c., although we may do with less) of gastric contents, to which a few drops of an alcoholic solution of phenolphthalein solution are added, a decinormal solution of caustic soda is added drop by drop from a graduated pipette until a red color appears and remains. The figure which is expressed by the amount of decinormal soda solution used in 100 c.c. represents the degree of total acidity of the gastric contents.

The normal values of total acidity usually vary between 20 and 60. If constant values below 20 are found, there is subacidity. If upon repeated examination values above 70 are noted, we are dealing with hyperacidity.

Hydrochloric Acid.—Of the many methods advised for the quantitative estimation of HCl I shall describe only the one proposed by myself which, among other advantages, possesses that of great simplicity, and has shown itself, both in my hands and in those of others, to be absolutely reliable.

To 10 c.c. of gastric contents, a few c.c. of a concentrated $CaCl_2$ solution are added, and after the addition of a few drops of an alcoholic phenolphthalein solution the mixture is triturated (as above) with a decinormal soda solution until a permanent red color appears. Then, about 15 c.c. of gastric contents are mixed in a dry vessel with about 1 gram of dry powdered $CaCo_3$, and, after stirring, are filtered through a dry filter, 10 c.c. of the filtrate are measured off, and the CO_2 which has formed is evaporated by a current of air, when $CaCl_2$ solution and some phenolphthalein are again added, and the acidity is calculated by means of a decinormal soda solution.

If the sum of the decinormal soda solution obtained in this tritration (calculated upon the basis of 100 c.c. of gastric contents) be subtracted from the number of c.c. required in the first tritration, the difference corresponds to the amount of normal soda solution required for the neutralization of free HCl present in 100 c.c.

of gastric contents. If the reaction for volatile fatty acids and lactic acids (see above) is negative (as is the case in the overwhelming majority of instances), the figure obtained corresponds to the amount of HCl. This figure multiplied by 0.00365 gives the percentage in grams of HCl in the gastric contents.

If organic acids be present, 10 c.c. of gastric contents are again measured and shaken in a beaker with about 50 c.c. of ether; the ether is then poured off, and this shaking and pouring off of ether is repeated five times. If the acidity of the residue of the stomach contents be then determined the difference shows the amount of the organic acids. This is subtracted from the total acidity previously obtained, and the remainder equals HCl.

FERMENTS

The presence of digestive ferments (pepsin and lab) may greatly assist us in the diagnosis of achylia (see below).

The test for *pepsin* consists in placing 10 c.c. of filtered gastric contents in each of two beakers, and also a flake of washed blood fibrin; to one of these a few drops of diluted HCl is added, and both beakers are placed in an incubator at blood temperature (98.6° F.). If, after several hours, the flakes are still present, in spite of repeated shaking of the beaker, pepsin and pepsinogen are absent from the gastric contents.

To determine the presence of the *lab ferment* about 10 c.c. of raw milk are mixed with 2 to 5 drops of gastric contents. Lab coagulation either occurs at once, or (at the temperature of the body) after a longer period. If the lab ferment is eventually absent, its prior stage, lab zymogen, may be recognized by adding 2 c.c. of a $CaCl_2$ solution to the previously mentioned mixture.

DYSPEPSIA

We employ the designation, dyspepsia, which in common parlance means "deficient digestion," to only a limited extent in that we restrict it to gastric digestion. But, even here, it is still further limited in that we exclude from this category organic diseases of the gastric walls which give rise to digestive disturbances.

In the term "dyspepsia" we include only those alterations of the stomach which are not due to an organic affection. It is true the ordinary conception of dyspepsia is decidedly more extensive, for we speak of dyspeptic difficulties when referring to abnormal sensations in the stomach without an anomaly of gastric activity being conjoined to them. This is especially true of so-called "nervous dyspepsia" (see below), the especial characteristic of which is that gastric digestion is quite normal. The contradiction in this is quite obvious, and although the designation which has become firmly rooted in our language is very difficult to displace, I nevertheless believe it to be correct in a systematic description to speak only of such conditions as dyspepsia in which gastric digestion is, in fact, disturbed.

By this classification, cases of dyspepsia are greatly limited. Since the gastric mucous membrane is too sensitive to retain for any length of time contents which have been subjected to decomposition without

reacting in a short time by irritative processes, i. e., by catarrh, only acute conditions belong in this category.

Under dyspepsia, therefore, and in a restricted sense, we consider only acute dyspepsia, i. e., the digestive disturbances which the busy physician meets with daily, which are due to the ingestion of tainted food or to generators of fermentation. As pure dyspepsia, without organic implication, these disturbances will be considered only in their initial stages, that is, before a consecutive catarrh has as yet been produced.

To determine that only acute dyspepsia, and not gastritis, is present, an investigation of the gastric contents is always necessary. The presence of products of decomposition, especially organic acids, volatile fatty acids, and lactic acid is not sufficient for this purpose. For even a primary gastritis, usually in consequence of anomalies of secretions, causes an abnormal change in the ingesta which become subject to acid fermentation. True dyspepsia, however, is favored by the absence of decided amounts of mucus, which, in acute gastritis, is always present in the gastric contents, especially in the later stages; a microscopic examination permits the recognition of numerous leukocytes and epithelial cells.

The effect also of a single, thorough gastric lavage, which is the best and most certain *therapeutic remedy* in acute dyspepsia, aids in the diagnosis; for if, after this little operation, and without other remedial measures, restoration to health is immediate, we may feel assured that the gastric mucous membrane was not implicated, and that the primary affection consisted in a decomposition of the gastric contents.

NEUROSES OF THE STOMACH

Neuroses of the stomach, with comparatively rare exceptions, are purely idiopathic. As a rule, they are caused by a general anomaly of constitution, or, reflexly, are dependent upon disease of another organ. In these cases, strictly speaking, we are therefore dealing only with a symptom of another disease which affects the stomach; the disturbances of gastric activity are, however, frequently so prominent and characteristic that they dominate the clinical picture, and for this reason alone they merit a comprehensive description.

The most important etiologic factor is neurasthenia, next hysteria, as well as chlorosis and other anemic conditions, diseases of the sexual apparatus (especially in females), wandering kidney and affections of the intestines, and these by no means exhaust the series of diseases in the course of which functional disturbances of the stomach appear. As a rule, they occur secondarily in cerebral and spinal cord affections, in pulmonary tuberculosis, in peritonitis, in severe renal disease, etc. Nevertheless, in making a diagnosis, we must consider their occurrence in very different diseases.

SENSORY NEUROSES

Anomalies of the Sensation of Hunger.—These consist in a decrease (*anorexia*), an increase (*bulimia*), and perversions of the normal appetite (*parorexia*).

Anorexia, which is a distinguishing symptom in almost all gastric diseases, may also be autochthonous, and here, above all, psychical influences such as care, sorrow, pain, disgust, etc., which dispel the sensation of hunger for a longer or shorter time, play a part. Naturally, before making the diagnosis, the existence of an organic gastric affection must be excluded.

Parorexia, in which there is a tendency to eat improper things, such as sharp condiments, vinegar, iron filings, chalk, etc., while natural foods are declined, occurs particularly in women who suffer from chlorosis or who are pregnant, but these abnormalities may also occur in other hysterical and neurasthenic persons. The treatment is that of the fundamental condition.

In *bulimia* we are dealing with a pathological augmentation of the normal sensation of hunger. This varies extraordinarily under different physiologic conditions, and, particularly in growing persons, is frequently intensified; and, since food not only serves to maintain the healthy condition of the body but also to promote growth, we can only regard excessive increase of the appetite as pathologic. This is shown by an abnormal hunger which appears soon after profuse amounts of food have been taken, and, besides the unconquerable desire to eat, it is accompanied by numerous distressing symptoms. Among these are lassitude, numbness of the head, sometimes actual headache, specks before the eyes, tinnitus aurium, general sweating, and tremor as well as general weakness. In some patients local abnormal sensations, as in the region of the stomach, are prominent; these are sometimes designated as pressure, sometimes as actual pain.

As the sensation of hunger is produced by direct action upon the hunger center (central disease and chemical irritation of the center by an abnormal condition of the blood) as well as by reflex causes which may originate from the periphery, it is evident that the causes of bulimia may be extremely numerous. Besides neurasthenia the following must be mentioned: Organic diseases of the brain and psychoses, Graves's disease, diarrhea, helminthiasis, menorrhagia, pregnancy, diabetes mellitus, etc. Those cases of bulimia are especially interesting which depend upon other functional diseases of the stomach, particularly upon hyperacidity (see below) and, above all, upon hypermotility (see below); in the former case it is apparently the abnormal irritation of the hyperacid gastric juice, in the latter the abnormally rapid emptying of the stomach after the ingestion of food, whereby a stimulation of the hunger center is caused centripetally.

The *therapy* of bulimia, on account of the great difference in the etiologic factors, must naturally be adapted to the underlying affection. The measures in the individual case are indicated in what has been previously said; in fact, in the case of helminthiasis, diarrhea, diabetes mellitus, and menorrhagia, a rational treatment of the underlying affection will in most instances suppress the bulimia, or, at least, decrease it. In hypermotility of the stomach it is wise to administer small quantities of food very frequently, and to alternate with fluid and solid food. In all cases in which there is hyperirritation of the nervous system, and these form the majority, besides the general régime suited to this, the administration of preparations of bromin as well as belladonna and similar remedies is of value.

Gastralgia (*cardialgia, gastrodynia*).—This affection which, under the picture of gastric crises, forms such a prominent symptom in tabes dorsalis and other central diseases, may also appear in consequence of the above mentioned affections as a substantive disease. Yet, upon the basis of my experiences, the cases in which typical and marked attacks of pain in the gastric region are observed, may only in an insignificant percentage be considered as gastric neuroses; even then the diagnosis is extremely questionable, for the positive exclusion of cholelithiasis and gastric ulcer (sometimes also panereatic calculi) is very difficult, and when we consider the great frequency of the two first mentioned affections, we had better, in a doubtful case, incline to these, and adjust the treatment in conformity.

Gastralgias of moderate degree, which cannot be referred to an organic affection, are, on the other hand, quite frequent. In these cases the pain or sensitiveness is referred to the entire epigastrium, and this circumstance, as well as the patient's frequent statement that the pains are lessened by pressure, is of value in the differential diagnosis between ulcer and cholelithiasis; in regard to ulcer, the independence of the quality of the food and of the time elapsed since the last ingestion of food also aids in the diagnosis.

Gastralgia is not infrequently combined with *hyperesthesia* of the gastric walls, which is particularly observed in anemic and nervous individuals, and which shows itself by a more or less decided sensitiveness directly associated with the intake of food. This sensitiveness may reach a very high degree, and cause severe irritative symptoms, such as convulsions and cataleptic spasms.

The main point in the treatment is to quiet the nervous system. Besides other measures, the constant current may be useful.

MOTOR NEUROSES

The disturbances of motility of the stomach consist in an increase or diminution of this function, affecting the entire stomach, or only the cardia or the pylorus. There are, however, pathologic pictures which are diffi-

eult to explain otherwise than as a eombination of irritative and depressing influenees.

Atony (insuffieieney) of the stomaeh, whieh appears so frequently in organie disease of the stomaeh (dilatation and gastreetasis), is one of the most important funetional disturbanees; it often oeeurs in anemia and other nutritive disturbances without demonstrable anatomie change in the gastrie walls. In this eondition we are dealing with atony of the museulature as well as a deerease in its elastieity and eonsequent impairment of the property of propelling the ingesta from the stomaeh at the proper time. As the abnormal tension of the gastrie walls whieh results is usually only a transitory extension of the organ, I shall omit its eonsideration at this point, and refer the reader to the exeellent artiele by Riegel.

Insuffieiency of the Cardia.—While flaeeidity of the museulature of the pylorus, eausing an insufficieney of the pylorus, has only been deseribed in isolated eases as a neurogenous affeetion (Ebstein), *insufficiency of the cardia* is an anomaly whieh may frequently show itself under different elinieal pietures.

Where there is a moderate degree of atony and a simultaneous tendeney to voluminous eolleetions of gas whieh are sometimes inereased by deeided swallowing of air (Quineke), we also observe *eructation* (ruetus, eruetatio) of gases, whieh may vary in intensity, and be transitory or habitual; in the latter ease this becomes very distressing to the patient. As the same symptom is eonspieuous in almost all organie gastrie affeetions, errors in diagnosis can only be avoided with eare. Besides hysteria, a eireumstanee in favor of the purely nervous eharaeter of the affeetion is that the gas which is diseharged is odorless and tasteless, and the eruetation may voluntarily be suppressed by the impulse of the will. Besides general tonies, the *treatment* must be designed to influenee the psyehieal element, and we must endeavor to strengthen the will power. Oeeasionally gastrie lavage is of use.

Another typieal elinieal pieture, whieh is attributable to ineontinenee of the mouth of the stomaeh, due not only to atony of the eardia but also to a flaccidity in opening and to antiperistalsis of the esophagus, eonsists in *rumination* (ruminatio, meryeism). In persons affeeted by this eondition, the ingested food rises from the stomaeh a short time after it has been swallowed, and returns to the mouth (*regurgitation*), where it is again ehewed and swallowed. This proeess usually oeeasions no disgust, and since regurgitation, as a rule, oeeurs soon after eating (rarely later than an hour), the food has no sour, or even unpleasant, taste. Many patients are unconseious of any disagreeable sensation, while others are keenly alive to their repulsive eondition. Not rarely they are able to suppress the regurgitation whieh they have voluntarily produeed, but only temporarily, for this afterward recurs to an increased extent. I know several patients who have had this affeetion for deeades, and who can pre-

vent regurgitation by lying down immediately after their meals, but if, after some time, they rise, the regurgitation at once appears. The circumstance that psychical alterations, fatigue, menstruation, etc., cause an increased and more frequent appearance of the affection is in favor of its nervous character.

The *treatment,* in the main, must be directed toward increasing the energy of the will and suppressing the pathologic process. Gastric lavage should be tried as well as preparations of bromin. Unfortunately, we rarely succeed in bringing about a cure.

So-called *nervous vomiting* may also be included in this class of affections. Vomiting is caused by increased muscular activity, but a paroxysmal contraction of the diaphragm and of the abdominal muscles only causes immediately a compression of the stomach, that is, an expulsion of its contents, and it is unquestionably true that the forcing of the stomach contents upward occurs all the more readily the less resistance there is on the part of the cardia. Nervous vomiting is characteristic because of the particular ease with which it appears (prodromes, especially nausea, are frequently absent), and it is obvious that flaccidity of the cardia plays an important rôle. This affection is prone to occur in hysterical persons, also in chlorotics, more rarely in neurasthenics. The idiopathic form of nervous vomiting described by v. Leyden, which is characterized by its periodic appearance (periodical vomiting), must be here included. Nervous vomiting may exist for years without the nutritive condition of the patient suffering.

[Regurgitation of food due to stenosis of the esophagus, especially at the cardia, may be mistaken for vomiting. This is especially true of cardiospasm. General nervous conditions may be the cause of the cardiospasm, and at the same time a nervous dyspepsia or a gastric ulcer may exist. A close attention to the character of the symptoms, especially of the act of so-called vomiting and an examination of the expelled food, will make the case plain.—Ed.]

In the diagnosis, an unusually large number of general affections and diseases of the most varied organs, intoxications and autointoxications, as well as organic gastric affections in which vomiting is a symptom, must be excluded, and furthermore, particular stress must be laid upon the etiologic factor of hysteria. Besides combating hysteria, local treatment of the stomach by lavage or irrigation and electricity are in place.

Hypermotility.—Hypermotility is a condition in which the motor activity of the stomach, by means of which its contents are propelled into the intestine, is increased. In consequence the stomach is empty during a time in which, normally, ingesta should still be present. Whether insufficiency of the pylorus also plays a causative rôle cannot be determined. Hypermotility is by no means rare. It occurs particularly in neurasthenics, and shows itself by the appearance of bulimia (see above). In

the diagnosis, the proof that the stomaeh one hour after the Ewald, or three hours after a Leube, test-meal is empty or eontains but few particles of food is neeessary. In regard to treatment, see that indicated for bulimia.

Peristaltic Unrest (*tormina ventriculi*).—This anomaly is distinguished by inereased peristaltic movement of the stomaeh (and frequently also of the intestine), and is accompanied by more or less loud rumbling which, with a thin abdominal wall, is distinetly audible. In making a diagnosis stenosis of the pylorus, above all, must be exeluded. The *treatment* consists in the use of sedatives; oceasionally lavage of the stomach acts favorably.

Spasm of the Cardia.—In contrast to the descriptions of this occurrence whieh are variously given, we must fully understand that the isolated spastic closure of the cardia may, primarily, cause diffieulty in deglutition. We therefore have the symptoms of a deeply situated stenosis of the esophagus. This anomaly is purely functional, particularly in hysteria, but it also occurs in marked anemia without other nervous irritative phenomena. It may run its course with or without pain, it may oceur paroxysmally, particularly in eonnection with the act of deglutition, or be permanent. Cases have been described in which the spasm had existed for years, and had given rise to a sac-like dilatation of the esophagus above the stenosis (Leichtenstern, Dinkler). Whether, however, in these eases an uleer of the cardia did not originally exist would be very difficult to deeide. For the diagnosis, examination with the gastric sound is deeisive. Upon its introduction, if a spasm at the height of the cardia exists, resistance is met with which, as a rule, is readily overcome. This proeedure may be aceompanied by pain or occur without it. In the absence of pain the diagnosis is easy. In a differentio-diagnostic respect, earcinoma, in the usually youthful patient, need scareely be eonsidered. But with a painful course of the affeetion, care must be taken not to confound the condition with uleer of the cardia. The successful introduction of the sound would exclude the latter, while, on the other hand, treatment similar to that for ulcer is to be instituted.

Besides general remedial measures, sounding the esophagus is indieated. Sometimes a single sounding is sufficient. If this is not the case it is well to apply cocain by moistening the lower end of the tube with a solution; or, still better, after introducing the gastric tube to the depth of the cardia, a few cubie centimeters of a not too concentrated cocain solution may be poured into the tube, which is allowed to remain *in situ* for a few minutes.

Spasm of the Pylorus.—This frequently occurs in organie diseases of the stomaeh, and is said to be also purely functional, partieularly in hyperacidity. The consequences are stagnation of the ingesta and tympany of the stomach, and the latter may be particularly severe *when combined with spasm of the cardia. Asthma dyspepticum,* so designated by Henoch, is due to a combination of this kind.

NEUROSES OF SECRETION

The anomalies of secretion are less numerous than those of sensation and motion. They consist in a decrease or increase of the secretion.

Achylia Gastrica.—While the achylia observed in connection with carcinoma and chronic gastritis depends upon an atrophy of the gastric mucous membrane, we observe in neurasthenic, and especially in anemic, persons, a decrease or complete absence of the secretory activity of the stomach without a causative anatomical change, and whether or not due to a purely functional disturbance is doubtful. Martius quite properly states that the ready vulnerability of the mucous membrane, so obvious in cases of this kind upon the introduction of the tube, raises the suspicion of the existence of slight degenerative or inflammatory processes. Besides, the subjective symptoms of achylia affecting the stomach are, as a rule, few, and their action upon the entire nutritive condition is slight, or may be wholly absent, for the absence of gastric digestion with intactness of the motor function is not of importance in utilizing the ingesta.

A *diagnosis* of achylia can only be made when repeated examination of the gastric contents, possibly after a previous irritation of the mucous membrane by the introduction of a salt solution, shows an absence of acid reaction or only very low values of the total acidity (at most ten or less). In the differential diagnosis carcinoma and gastritis must be excluded. In the *treatment,* besides general strengthening therapy, the absent secretion must be substituted for by the administration of hydrochloric acid and pepsin at every meal, and the maintenance of the motor activity of the stomach must be provided for. The former, as has been remarked, is unnecessary for the sufficient utilization of the ingesta. There is, however, no doubt that by the absence of hydrochloric acid in the stomach there is danger of the further development of pathogenic microörganisms which have gained entrance there, and this danger may be averted by the administration of hydrochloric acid.

Hyperacidity (*hyperchlorhydria, gastroxynsis*).—Hyperacidity represents one of the most important disturbances of function of the stomach, and it may also be combined (see below) with hypersecretion (gastrorrhœa acida). It is chiefly noted in young, anemic individuals, particularly chlorotics, and also in neurasthenics, but by no means exclusively in these conditions. Psychical irritation is unquestionably an important etiologic factor, but it is also observed in persons whose nervous system is, in general, quite normal. Of organic gastric affections, which we shall not minutely consider here, the anomaly is most often associated with ulcer. We find it, too, in motor insufficiency of the stomach, in gastrectasis, in gastroptosis and purely functional atony (see above), probably due to the abnormally long presence in the stomach of the ingesta which causes the secretion. Cases of hyperacidity have also been described (acid gastritis)

in chronic gastritis which, as a rule, runs its course with a decrease in gastric secretion, and in the amount of acid. On account of the extreme difficulty of excluding these organic affections, we must admit that absolutely certain cases of purely functional hyperacidity are by no means frequent.

The subjective symptoms, combined with a normal appetite, are pyrosis, which may be increased to gastric pain, pressure in the gastric region, and acid eructations. Vomiting of decidedly acid masses is rare; thirst and bulimia are frequent. These symptoms appear in very different degrees without a parallel degree in the amount of acid. The sensitiveness of the gastric mucous membrane to the irritative action of hydrochloric acid is apparently very variable in the same and in different individuals, and the relation in time of these difficulties to the intake of food shows many variations. Sometimes they are nearly continuous with occasional exacerbations attributed to the food consumed, at other times they occur only paroxysmally after meals. Sometimes also certain foods which are invariably not well borne are responsible for the difficulty.

The *diagnosis* of hyperacidity depends upon an examination of the gastric contents. If, upon repeated examination, we find an amount of 0.25 per cent. of HCl or more, hyperacidity is proven. If upon the absence of organic acids the total acidity is more than 70, the same condition is proven. The circumstance that particles of bread are less broken up than normally is also characteristic of a high degree of acid. It must also be borne in mind that, just as under physiologic conditions, variations in the amount of acid frequently occur, yet the diagnosis of hyperacidity is not superseded by the fact that occasionally normal amounts of acid are found.

While the proof of hyperacidity, as a rule, is simple, the exclusion of an organic disease of the stomach and the proof of a purely functional disturbance combined with hyperacidity may be most difficult. Here, in the first place, gastric ulcer must be mentioned. On account of its great frequency, and that, as a rule, it runs its course with hyperacidity, and also that, in this affection, the symptoms usually appear some time after eating, we cannot be too careful in the differential diagnosis. When there is well developed gastric pain besides the other mentioned symptoms, in my experience a purely functional hyperacidity should be excluded and the treatment should be based upon the hypothesis of a gastric ulcer. If positive factors for the development of an acid gastritis have been proven, in the differential diagnosis we must also consider this affection. Contrary to the reports of some authors, the absence of mucus in the evacuated masses favors the assumption of the purely nervous character of the malady, if the evacuation have occurred at the usual time after the test-meal, for the mucus secreted in consequence of catarrh is very rapidly digested by the acid gastric juice, which has a strong, peptonizing action. More signifi-

cant, on the other hand, is the fact that toward the end of digestion, upon microscopic examination of the remnants in the opening of the tube, large numbers of cell nuclei are found, since, normally, but few nuclei of this kind are present. Another circumstance that favors an existing catarrh is that upon withdrawal of the tube from an empty stomach adherent particles of mucus containing large numbers of leukocytes are found.

The views in regard to the best *treatment* of hyperacidity have undergone decided change. The fact that an increased amount of HCl in the gastric contents unquestionably disturbs the saccharification of the food-containing starch by the salivary ferment which is swallowed made it appear rational to limit starchy food in the diet of the patients suffering from hyperacidity (to the utmost), and to place them upon animal food. Experience has, however, shown that a diet of this kind does not always lessen the difficulties, but not infrequently they increase, and this coincides with the fact that objective investigations of the gastric contents show no decrease in the amount of acid. In the first place, a meat diet is generally not well borne by patients with gastric disease, and, above all, a diet rich in albumin causes a greater secretion of acid (Hemmeter, Fleiner, Jürgensen and others). It is, therefore, rational to prescribe for the patient a mixed diet which possesses the property of combining acids, which increases acid secretion to the slightest degree, and is as little irritating as possible. These indications are best fulfilled by milk, and by milk rich in fat, since fats have an extremely slight influence upon the gastric juice. If milk is declined, or not well borne, as is unfortunately often the case with these patients, lime water may be added to it. Soft, pappy rice, maizena, stewed calves' brains, sweetbreads, etc., are given. Toasted wheat bread, and tea with a plentiful addition of cream, are likewise permitted. We also prescribe soda bicarbonate, magnesia (hydrated), Carlsbad salt and other alkalies, besides narcotics (especially belladonna). In some cases there is a favorable influence from takadiastase, whose saccharifying property is less disturbed by hydrochloric acid than that of ptyalin. In addition, stimulating measures are not to be neglected.

Hyperacidity is a very stubborn disorder, and we must, therefore, be guarded in the prognosis.

Hypersecretion (*flow of gastric juice*).—This affection is characterized by an abnormally profuse secretion of the gastric juice, which occurs without the stimulation of the ingesta. It may be continuous or periodic, and may run an acute or chronic course. The amount of HCl of the secretion may be increased (gastrorrhœa acida) or normal. In the former case the symptoms are those of hyperacidity; but, even when HCl is not increased, the patients complain of pyrosis and acid eructations. Vomiting of a more or less profuse, watery, acid fluid while the stomach is empty of food is characteristic of the condition.

The diagnosis depends upon the last mentioned symptom and upon

the results obtained by the stomach-tube while the stomach is empty; that is, whether by this procedure large amounts (100 c.c. to 1 liter) of an almost clear fluid having an acid reaction and containing HCl and pepsin are evacuated.

The treatment must be directed to the removal of the probable underlying affection, neurasthenia, anemia, etc.; the preparations of bromin, narcotics (codein, belladonna, cocain, etc.), are in place as well as irrigation of the gastric mucous membrane.

NERVOUS DYSPEPSIA (GASTROSIS)

Among the gastro-neuroses, so-called "nervous dyspepsia" occupies a peculiar position. Leube, who accurately portrayed the clinical picture after it had been described by others, particularly by English authors (Beard and Rockwell), defines it as a pathologic condition in which subjective symptoms of chronic dyspepsia, analogous to those of chronic gastritis are present, but in which objectively demonstrable anomalies of activity, as well as organic changes of the stomach, are absent.

It is evident that, in a pathological condition the basis of which is subjective difficulties, the negative results of investigations of the anatomical and functional disturbances of the stomach create a difference of opinion among authors in regard to the definition and the limitation of the affection, and that many deny that there is any justification for considering a symptom-complex of this kind as a disease, *sui generis*. In fact the number of authors who are opposed to the pathologic conception of "nervous dyspepsia," in Leube's sense, is quite considerable. We would digress too far, and in entire opposition to the character of this article, were we minutely to consider the comprehensive literature of this subject. Some of these explanations bear a remote date, and it may be assumed that not a few of the opponents of "nervous dyspepsia" have, in the course of time, made subsequent observations, and have changed their former opinions.

According to my experience which coincides with that of others, there is no doubt of the existence of a clinical picture as described by Leube and others. Far be it from me to maintain that, in such cases, an organic change can always be excluded. We are unable, however, to demonstrate it, and we may often convince ourselves that no organic disease known to us is the cause of the affection. Shall we therefore deny the existence of this clinical picture? With as much right as we could eliminate many other affections, such as migraine, neuroses of the heart and of the intestines, flittering scotoma, pruritis, etc., from pathology. For in these conditions also we are unable at present to detect any anatomical anomaly which is certainly the cause of the symptoms or to exclude it. The justification and the necessity of limiting "nervous dyspepsia" as a disease, *sui*

generis, as well as the previously mentioned functional disturbances, simply depend upon experience, in consequence of which the symptoms mentioned and those still to be discussed appear, and must be considered, as the expression of this affection of the stomach.

Objections have been raised to the description of the clinical picture, as well as to its name. This is quite reasonable. Since objective disturbances of digestion are absent in the disease it would naturally follow that the name "nervous dyspepsia" would provoke discussion. It might also be added that the nomenclature does not correspond to the actual conditions, since the dyspeptic difficulties are, without exception, to be attributed to an implication of the nervous apparatus. In many, perhaps in a majority of the cases, this origin may be at once admitted; namely, for those in which the disease depends upon neurasthenia, hysteria or organic disease of the nervous system, or where it appears as a reflex neurosis, particularly from the sexual apparatus. Even where the causative factor is looked for in intoxications or infections, such as the abuse of nicotin, alcoholism (here it is true there is usually chronic gastritis), syphilis, malaria, etc., which usually exert a damaging influence upon the peripheral nerves, we are justified in ascribing to the primary cause the development of the gastric symptoms. On the other hand, it must be remembered that the symptom-complex peculiar to this group also appears in consequence of anemic and general debilitative conditions, such as chlorosis, the puerperium, improper and insufficient food, etc. It would seem far-fetched to look for the cause of the morbid sensations in the nervous system. This would be more obvious in an insufficient nutrition of the parenchyma of the stomach which is undoubtedly present, which, however, as the actual causal factor, has as yet shown no gross pathologico-anatomical changes of the organ. For this reason, the designations neurasthenia dyspeptica or gastrica (Burkart, Ewald) are not to be regarded as improvements; for, as v. Leyden quite properly remarks, the chief disturbances are not in the stomach, as should be the case. Of the other names which have been proposed, the best appears to be "combined gastroneurosis," which, it is true, is somewhat complicated, and does not emphasize the sometimes non-nervous nature of the affection.

Perhaps it would be wise to choose a more indifferent designation, which would, however, permit us to recognize that we are dealing with a functional anomaly of the stomach. As such I propose the name: *Gastrosis*. The designation nervous dyspepsia has become so firmly rooted in our minds that it will not readily be eradicated, but the name is of subordinate significance provided we are unanimous as to what it signifies.

Among the etiologic factors which have been mentioned there is one which is important because of its comparative frequency. This is an abnormal state of nutrition due to the fact that the periods between the

individual meals are too long. There are some persons who, like dogs, take only once a day a meal which really deserves the name, and yet they continue in the best of health. In spite of the latter circumstance, there can be no doubt that this habit is irrational. It is contrary to the laws regulating the function of the stomach as a reservoir of food, and it cannot be an advantage to the stomach to remain empty for several hours during the day and then suddenly to become excessively loaded. The effect of this is particularly noticeable in persons who do not voluntarily or from habit make these long pauses between meals, but are forced to it by the stress of circumstances. This occurs in very busy physicians, particularly surgeons, in merchants, in lawyers, etc. According to my experience, this category of occupations furnishes the main contingent of the cases of gastrosis. The circumstance that the digestive disturbances of the patients improve when they change their mode of life and take more frequent but smaller meals, is proof of the etiologic importance of this improper mode of nutrition.

The *symptoms* of nervous dyspepsia, as already stated, are in the main subjective. The patients complain chiefly of a feeling of pressure and distention in the gastric region, and of eructations which sometimes become exceedingly severe and loud. The appetite, as a rule, is disturbed. The patients have more or less repugnance to food, and not rarely complain of nausea. Tendency to vomit and actual vomiting also occur, but not so frequently as in gastritis. The loss of appetite in some cases depends upon autosuggestion. The patients fear that by eating their distress will be increased, and therefore refrain from food as far as possible. On the other hand, even bulimia occurs; the patients then are tormented by a voracious appetite which causes them to crave food. As a rule, as soon as they begin to eat, the bulimia not only disappears, but also all desire for food. They can only take small amounts, and are unable to satisfy the existing hunger. Perverse anomalies of taste are also frequently observed. Acids and other foods difficult of digestion are especially preferred, and in the circumstance that, after their use, the preceding discomfort often lessens, this clinical picture differs from other organic affections.

Among the abnormal sensations which originate from the gastric region, besides fulness and eructation, pyrosis must be mentioned. This symptom, which is an almost invariable accompaniment of actual dyspepsia, of "sour stomach," is not always found hand in hand with an increase in the acidity of the gastric contents, nor need this be the case in nervous dyspepsia. There are, however, instances in which the degree of acidity exceeds the normal limits.

Actual pain in the stomach is but rarely complained of, and, even then, it is not the pain upon pressure which is so characteristic of ulcer. The gastric region may be sensitive to the pressure of palpation, but often this

is only an indefinite, disagreeable feeling, which is also observed in other sensitive patients whose stomach and other abdominal organs are perfectly normal.

The condition of the bowels varies. Constipation exists in many cases, as in other chronic gastric affections. Diarrhea is rare, and often no anomaly is present.

The disturbances of the general condition may be manifold and correspond to the varied etiologic bases of the affection. From the symptoms of neurasthenia (such as numbness in the head, lassitude, depression, hypochondriasis, even melancholia, on the other hand frequently slight irritability, cardiac palpitation, vertigo, etc.) it is not always easy to decide whether we are dealing with coördinated symptoms of the same cause of the disease or with the sequels of the gastric affection.

The general nutrition may also vary according to the causal factor. In most cases the patients are well nourished, and there is no external sign of suffering. But we also see patients who are hollow-eyed and more or less emaciated with no malady save one of the stomach to account for the disturbance. The history then reveals that the emaciation is due to insufficient food. These patients, partly from fear of distress, partly in obedience to unwise professional advice, have for a long time taken only a minimum of food, and have actually undergone a starvation cure. It cannot be wondered at that, in consequence of this limited diet and the resulting loss of resistance of the walls of the stomach, the symptoms instead of decreasing have been decidedly aggravated. These are the most hopeful cases for treatment; the physician who clearly recognizes the affection and acts upon this knowledge, helped by the previous error, may, with ease, bring about what seems a miraculous cure.

In the *diagnosis* of nervous dyspepsia, which should be made with great care, we must first decide whether the complaints of the patient are actually attributable to gastric activity, or whether the difficulties referred to the stomach are not due to a disease of other parts of the organism, or whether they do not at least owe to it their origin. In this connection we cannot be too skeptical, since such so-called dyspeptic symptoms as were emphasized above may occur in a number of different affections which cannot be minutely described at this point.

The presence of well developed neurasthenia or hysteria, and the proof that these anomalies existed prior to the dyspeptic symptoms, are of importance in the diagnosis. It is obvious that these will aid us only in the diagnosis, as neurasthenic and hysterical persons may also suffer from organic disease of the stomach, and as gastrosis also occurs in persons who, in the main, show no functional disturbances of the nervous system.

The coincidence of dyspeptic disturbances belonging to the symptom-complex of nervous dyspepsia, with functional disturbances of the heart without organic disease, is also important in diagnosis. Cardiac palpita-

tion and irregularity of the cardiac action in connection with gastric affections are, therefore, symptoms which must be considered in the diagnosis.

The report of the patient that the digestive symptoms vary, that, instead of the usual disturbances, there are times when the disease seems to have almost disappeared, particularly when the mind is diverted by pleasant company, may be of value. With Rosenheim I admit this fact, but it rarely occurs, and is not absolute proof that other gastric patients, for example, those suffering from chronic gastritis, occasionally will forget their dyspeptic disturbances when in interesting society, at the theatre, etc. Nevertheless this occurs, although less frequently.

The objective examination of the stomach is chiefly important because conspicuous anomalies are not found. I am of the opinion that the moderate grades of gastroptosis which prevail so commonly in women without giving rise to symptoms, cannot annul the diagnosis: Nervous dyspepsia.

In regard to the functional test, Leube, as remarked above, has laid special stress upon the fact that the motor function is intact. According to my experience, we must in general agree with him, and the cases in which there is a well developed slowing of motion should not be considered as nervous dyspepsia. Moderate grades of atony, or the presence of small remains of food two hours after a test breakfast and seven hours after a test-meal, belong to the same clinical picture, particularly if this condition is not constant. In testing the functions of the stomach these frequently great variations are characteristic, and this is true of a higher degree of secretion. In this respect I entirely agree with Boas and Rosenheim. It is true that while the degree of acid is usually within normal limits we occasionally note surprising variations of the acidity below and above the normal.

On account of this variability of the symptoms, and the difficulty of limiting exactly the clinical picture, we should never fail to consider the possibility of organic disease of the stomach before we make a positive diagnosis.

The condition may be mistaken for *gastric ulcer,* for this affection does not always present the well known classical symptom of typical pain occurring a short time after the intake of food, but it may develop without any pain, and an examination of the gastric contents may reveal no characteristic differences. If there is actually well developed pain, the case is not one of nervous dyspepsia. Marked tenderness and a drawing sensation in the gastric region, however, are observed, and the report that these sensations are not increased by external pressure but are rather relieved is decidedly against ulcer. The manner in which the sensitiveness of the gastric region shows itself upon palpation is important. If it be localized to a circumscribed area this favors ulcer, while a diffuse, painful sensitiveness of the entire epigastric region points to the nervous character of the affection. If hysteria is present we not rarely observe, especially

in thin patients, painful pressure areas upon both sides of the vertebral column. But a confusion of this previously mentioned pain upon pressure with simultaneous gastroptosis is not impossible.

Even if there is no marked sensitiveness of the gastric region, we cannot at once exclude ulcer, particularly when dealing with young anemics. As the examination of the gastric contents permits no positive conclusions, it is wise in cases of this kind to proceed as if ulcer were present, therefore, to prescribe absolute rest, fluid diet, and silver nitrate. If by this means the affection is not favorably influenced, the assumption of gastrosis is justified.

Gastric carcinoma may be excluded with certainty provided the dyspeptic difficulties have existed for a long time without any diminution of the body-weight. We must, however, remember that carcinoma may run a very slow course without producing symptoms other than those of dyspepsia, anemia, and slight emaciation. In elderly individuals we must be very cautious in the diagnosis of an affection of this character, existing for a short time, perhaps only for a few months. If well developed symptoms of neurasthenia or hysteria have preceded, they favor the diagnosis of nervous dyspepsia, although, of course, patients of this kind may be attacked by carcinoma. A normal hydrochloric acid reaction and the absence of lactic acid in the stomach contents are factors which decidedly favor nervous dyspepsia. Absolute certainty, however, can only be attained when regular weighings of the body, under sufficient nourishment, show no loss in weight.

Well developed *gastrectasis* presents such a characteristic clinical picture that this disease can scarcely be mistaken for another. But this may readily occur when the symptoms depend upon a simple *atony of the gastric walls.* Here definite conclusions are reached by the examination of the motor function. The proof that there is no stagnation of the ingesta, that, on the contrary, the stomach discharges its contents normally, excludes this anomaly. Naturally, we should not be content with a single examination, for, as has been stated, variations in the motor function occur in nervous dyspepsia. But the existence of this affection is not disproven if, transitorily, a prolonged retention of the ingesta in the stomach is observed.

Chronic gastritis must be specially borne in mind in the differential diagnosis, as the subjective symptoms of this condition completely coincide with those of nervous dyspepsia. The absence of a coated tongue, and the non-appearance of mucus in the vomit and evacuated gastric contents, particularly when the stomach is empty (see above), favor the latter. If gastritis is present, upon microscopic examination of the contents of the tube we find profuse formed elements, leukocytes and epithelium. The chemical examination of the gastric contents after a test-meal is not unimportant, for, as a rule, the secretion of HCl is deficient in gastritis. This

is, of course, not always the case, for in so-called acid gastritis, which is rare, it may even be increased. Therefore, in cases of this kind, the circumstance that the affection occurs in neurasthenics or hysterical persons, as well as the modification of the disease by treatment, is of significance.

The *prognosis* is always doubtful. By proper treatment many cases are favorably influenced and even complete recovery ensues. But it sometimes happens that, at the onset, decided improvement takes place, and the easily excited patients are enthusiastic regarding the result; then, however, relapses occur which affect the entire nervous system, and reproduce the former gastric disturbances. In other cases all endeavors favorably to influence the condition are without avail. These cases test the patience of the sufferer and the physician.

In *therapy* the psychical treatment is hardly less important than the somatic. It is true we will only rarely be successful if we represent to the patients that the affection is a very slight one, which may yield to a little exercise of the will. As a rule the patients resent a suggestion of this kind. It is in crass contrast to their discomfort, and, as a matter of fact, the mere determination not to be ill rarely suffices to cure the disease.

But there are instances, as stated before, in which a mere change in the diet, i. e., a return to the usual home living, is sufficient to restore the patient to health. These are, however, not cases due to neurasthenia or hysteria, but those which at the onset were very insignificant gastric difficulties, but excessive carefulness and a fear of the injurious effect of wholesome and plentiful food has limited the diet to such uniform and insufficient food that it has not conduced to improvement; hence, in consequence of imperfect nutrition of the walls of the stomach and of the entire organism, a steady aggravation of the dyspeptic symptoms has been brought about.

A., a manufacturer, aged 45, as a young man had disease of the left pulmonary apex, which, however, entirely disappeared and there is no trace of it at present. After this illness, the patient became extraordinarily careful and anxious about his health. The slightest deviation from the normal made him fear the onset of disease.

According to report, several years ago he began to have a sensation of fulness and pressure in the epigastrium after his principal meal, accompanied by eructations and constipation. Gastritis was diagnosticated, and the patient, whose appetite was always normal, limited to an extreme the quantity and quality of his food. By this means and the use of Carlsbad water, his condition is said to have at first improved, but gradually there was an aggravation, and, although pressure in the region of the stomach was no longer especially noticeable, the eructations continued, and to these vertigo, headache and palpitation of the heart were added. Appetite was lost, and not infrequently the monotonous diet caused repugnance. Nevertheless, the patient continued upon this diet through fear of injuring himself by a change.

The examination of the powerfully built, but pale and poorly nourished patient, revealed no anomaly of the stomach (total acidity upon repeated examination 30 to 60, Günzburg's test positive, motility normal) or any other part of the organism.

I explained to the patient that there was no disease of the stomach, nor, in fact, any organic affection, and that his symptoms were due to an injudicious mode of life, particularly to monotonous and insufficient food. I, therefore, advised him not only to take the "easily digested foods" but to return to his former favorite foods. No restrictions, whatever, in diet were necessary. The patient at first was somewhat incredulous but followed my advice and at once ate some peas with sour kraut and pickled meat. As the symptoms which he looked for did not appear he gained confidence, again ate and drank like a healthy man, and after three weeks presented himself to me completely cured. His weight steadily increased (about 21.9 pounds) and except for constipation, which still existed, all symptoms had disappeared.

Cases of this kind are, however, infrequent, and, particularly when dealing with nervous persons, treatment such as above described is not in order. Here, above all, it is necessary to tone up the entire nervous system. Bodily and mental rest, removal from the ordinary occupation, are usually the first requirements, but by no means always. Patients who have previously passed their time in idleness, with hypochondriae thoughts dwelling upon their stomach affection, should be advised to take up a stimulating occupation, strengthening and even fatiguing bodily exercises. But the diet must be regulated. In severe cases, particularly in debilitated women, complete isolation and rest in bed, at least for the greater part of the day, and forced feeding, will be beneficial.

In other cases a change of air, i. e., residence in pure fresh air (medium altitude in the woods) is sufficient. Special stress must be laid upon the stimulation of the skin by baths, massage and electricity. Above all, the diet must be regulated. Food difficult of digestion, and particularly that which causes flatulence, must be avoided, but the fact should be emphasized that the diet should be varied as much as possible, and the intervals between the meals are not to be too long.

Drug treatment, compared with these general dietetic rules, is of decidedly secondary importance, but it is frequently necessary to support the latter mode of treatment. Here stomachics such as rhubarb, tincture of quinin and bitters, iron and Fowler's solution, perhaps also quinin as a tonic, then sodium bromid and valerian, as well as various remedies for the usually stubborn constipation, are beneficial.

THE DIAGNOSTIC AND THERAPEUTIC SIGNIFICANCE OF SECRETORY DISTURBANCES OF THE STOMACH

By H. STRAUSS, Berlin

The significance in diagnosis and therapy of the secretory disturbances of the stomach is unquestionable, and is important from a scientific as well as a practical standpoint. This will be admitted even by those who hold the certainly justifiable view that the nosological significance of disturbances of secretion was greatly over-estimated in the first few years of the new departure in functional diagnosis of the stomach. But there is no reason for under-estimating it to-day. Though we must acknowledge from actual observation that a person absolutely without secretion in the stomach may not only live for years but even decades without showing noteworthy disturbance of nutrition, we know, nevertheless, that the result of marked secretory insufficiency of the stomach—as, for instance, diarrhea—may damage nutrition to a marked extent, and that, on the contrary, excessive secretion—continuous secretion of the gastric juice—by causing chronic under-nutrition, dryness of the tissues, and perhaps also withdrawal of chlorin may induce conditions which threaten life. It is not my object to describe in detail these conditions in which disorders of secretion play a rôle, but I shall consider secretory disturbances only in so far as they represent subjects for *diagnostic* and *therapeutic* consideration. If this manner of presenting the subject is *a priori* unfamiliar to the reader accustomed to regard definite clinical pictures as nosological entities, it certainly will facilitate the description, and the physician who understands also other disturbances of the stomach, their importance, and the methods for their recognition, will not be led by such a description to a too one-sided conception of the subject, or allow himself as a therapeutist to drift into errors of omission. As a rule, the careful physician will have already grasped the knowledge that, as a basis for the erection of pathologic pictures in gastric pathology, the clinical symptom-complexes which we meet to-day in gastric pathology frequently do not equal the pathologico-anatomical changes with which we have long been familiar. Following O. Rosenbach, almost all investigators in this realm have come to the conclusion that for the purpose of treatment the interests of our patient demand not only a thorough investigation of the symptoms of *anatomical* change (position, form, and size of the stomach, production of mucus and pus, superficial

lesions), but, in every case also an accurate study of the individual *disturbances in function.* In many instances our method of *treatment* will only enable us to reach the goal by a *test of function* (secretion, motility, etc.).

GASTRIC SECRETIONS

Among the different secretions of the stomach (hydrochloric acid, pepsin, lab-ferment, steapsin, water) the secretion of hydrochloric acid occupies the first place in our present *clinical* description, as the disturbance of other secretions, such as pepsin and lab-ferment, the secretion of steapsin, and of the diluting secretions, either is *so far* parallel with disturbances in the production of hydrochloric acid that for *clinical* purposes it requires special consideration only in particular cases, or, according to the existing state of our knowledge, it is to be regarded as of so little practical value that, for our present purpose, we need not enter upon its detailed description.

When we refer to a disturbance of secretion as an object for *diagnosis and treatment,* the first question that arises is in regard to the normal or pathologic limits of the secretion. This question can be no more accurately answered than we can determine by a simple, fixed formula the boundary between health and disease; for the relation of gastric juice secretion in individual persons not only varies within wide limits but also in one and the same individual at different times; just as in every other function, this has an intimate connection with the nervous system. This has been shown conclusively by the clinical study of cases and by experimental physiology—I refer here, above all, to the classical investigations of Pawlow. Moreover, investigation of the gastric contents in the individual case may reveal an extreme increase or diminution of gastric juice secretion without the person in question presenting the slightest subjective sign of disease. Regional differences in the realm of gastric juice secretion may also be recognized, as I demonstrated some years ago in my experiments at Giessen and Berlin. If the production of hydrochloric acid be taken as a test object for gastric juice secretion, in a stomach acting normally, one hour after taking a test breakfast, that is, three to four hours after the ingestion of a test-meal, we may expect to determine free hydrochloric acid in the gastric contents, and that in a test breakfast, as a rule, the total acidity will be not higher than about 60, and in a test-meal not higher than about 100. The lowest computation of total acidity in a trial breakfast under normal circumstances will rarely exceed from 35 to 40, and in a test-meal from 60 to 70.[1] I have here quoted figures, but, in spite of this, I must

[1] From what has been said it is at once evident that, if we desire to form an opinion regarding the secretory activity of a case, a single examination is insufficient, but repeated examination of the gastric contents is necessary.

premise that the determination of the total acidity represents only the final act in the test of the secretion of the stomach. We do not begin the examination of the heart with auscultation, neither do we introduce the test of the secretion with titration, but we necessarily assign the first place to close inspection of the gastric contents.

As I do not desire to become involved to any great extent with the question as to which tests are to be preferred for daily practice, I shall only state here that many roads lead to Rome. I, myself, am of the opinion that although the introduction of the test-meal has deepened our insight into the mechanism of gastric digestion, and that in many respects and even to-day, in a concrete case, it is sometimes calculated to enlarge our knowledge, nevertheless, in the majority of cases, the test breakfast suffices for a practical test of function, provided it is carried out *lege artis,* and its results are subjected to examination from all points of view. The last requirement must, however, be particularly emphasized if we assign to the test breakfast the position just indicated; and here it must be remembered that in some cases of hyperacidity, and under some circumstances after administering the test breakfast, a comparative test with a test-meal may be desirable, and also that there are rare cases of "perverted secretion" (Riegel).

Inspection.—*On inspection of the test breakfast,* the consistency of the sediment, which resembles flour soup, or purée without the appearance of coarser particles in the expressed contents, shows normal or increased gastric juice secretion, while a gross, fragmentary appearance in the evacuated gastric contents leads us to suspect insufficiency of gastric juice secretion. The "amylorrhexis," so designated by me (in contrast to amylolysis), is only a function of proteolysis, and, as such, an indication which may be well utilized in studying the secretion of the gastric juice. Any one accustomed systematically to regard the test breakfast from the point of view of the degree of "amylorrhexia" may, even from the macroscopic examination of the evacuated contents, determine the presence of subacidity and—although with less certainty—also assume the existence of hyperacidity. Under certain circumstances, the microscopic examination of the secretion may give us valuable aid. Thus, as a rule, in extreme subacidity, net-like formations are found which I some time ago described as "structure substance," because they consist of the adhesive structure—giving the histochemical reaction of albumin—in which granules of starch are embedded. Such structures are found particularly in "apepsia gastrica." Furthermore, in those cases in which leukocytes are admixed with the gastric contents, a high-graded secretory insufficiency may also show the retention of the protoplasm ring of the leukocytes which is absent in all those instances in which a more marked production of acid is present—according to my experience, even the presence of free hydrochloric acid may be unnecessary.

For practical purposes the methods which are to be considered in the

investigation of the products of secretion of the gastric mucous membrane have been detailed to a great extent in the article by Professor Leo, "Functional Diseases of the Stomach" (page 66). I shall not consume time with their description, but shall allow myself only a few critical remarks based upon personal experience. At the close of this article I shall enumerate a few methods not given in the previously mentioned treatise.

Test for Hydrochloric Acid.—For the test of *free* hydrochloric acid I see no reason to reject Congo paper, since this permits in the simplest manner the sufficiently positive recognition of its presence. The dimethylamidoazobenzol paper recently advised for this purpose by Einhorn I did not find superior to Congo paper when I conducted investigations about eight years ago in Riegel's Clinic. Only when Congo paper gives indefinite results, should the Günzburg reagent be employed. For the quantitative estimation of free hydrochloric acid I still advise dipping with Congo paper according to the processes of Riegel, Mörner and others—perhaps also the utilization of dimethylamidoazobenzol as an indicator. With Linossier's reagent, which also contains phenolphthalein and dimethylamidoazobenzol, I have not always obtained a definite result. The question in which cases free hydrochloric acid should be determined *quantitatively* can, in my opinion, only be decided in practice in the individual case, since the physician who frequently investigates the gastric contents learns in the course of time, from the combination of values for total acidity, from the Congo reaction, as well as from other signs in the gastric contents, what practical conclusions are to be drawn in the majority of cases. From my own experience I maintain this in regard to the determination of *combined* hydrochloric acid, and I have only rarely found a strict necessity for its quantitative estimation—at least for practical purposes. In the overwhelming majority of cases in which there is a deficiency of free hydrochloric acid and the absence of lactic acid, the comparative estimation of the proportion of total acidity and the degree of digestion in the evacuated contents enable us to form an opinion sufficiently clear for practical purposes as to the amount of secretion and the measure of the peptic labor of the stomach, provided we start with the assumption that when large quantities of combined hydrochloric acid are present in the evacuated contents, a good degree of digestion may, as a rule, be recognized, while this is absent when there are only slight quantities of combined hydrochloric acid. I cannot, however, strongly recommend the complicated methods which are advised for determining combined hydrochloric acid, as their adoption into practice is beset with great difficulty, and the exactness of their results is often not commensurate with the complexity of their use. If we desire to know the amount of secretion while excluding the phosphates, we may titrate with phenolphthalein and tincture of cochineal (see later), or Leo's process may be employed, or a trial may eventually be made with a method recently advised by Colmheim and Krüger. In my experience, however, the latter

necessitates the utmost care, and gives only approximate, not always absolutely exact, values for combined hydrochloric acid if, as I found when working with Dr. Cahn, at the same time, investigations are made of the gastric juice for free hydrochloric acid, utilizing Leo's process. The previous method, advised by v. Noorden and Honigmann, for "hydrochloric acid deficit," is simple but does not always give similar values in regard to the amount of combined hydrochloric acid in a test-meal at the height of digestion, as I determined after numerous investigations of the gastric contents for free hydrochloric acid with Leo's process, and with Congo titration; Ehrmann determines it at from 0.05–0.07 HCl—particularly in cases of stagnation with good secretion—and also higher values are observed.

Total Acidity.—The total acidity of the gastric contents, as is well known, is determined by the aid of phenolphthalein which reacts upon all three of the acid components of the normal gastric contents (free and combined hydrochloric acid as well as acid phosphates). For the determination of the "physiologically active" hydrochloric acid alone this property of phenolphthalein is unsatisfactory, especially in the cases in which, by a diminution of the production of hydrochloric acid, the amount of acid phosphate in proportion to the total quantity of the "physiologically active" hydrochloric acid increases. The acid phosphates, as I am in a position to prove, form no inconsiderable part of the total acids. In 14 investigations (according to Leo's process) I found that the total quantity of acid phosphates in the empty stomach, that is, in the secretion free from food, amounted in value to 7.5 (maximum 10, minimum 5); in 19 test breakfasts with free hydrochloric acid, an average value of 11 (maximum 15, minimum 8); and in 14 test-meals with free hydrochloric acid, an average value of 22 (maximum 38, minimum 9). Therefore, another indicator would be desirable which, without reacting upon the phosphates in the absence of lactic acid, would show exactly what acid factors of the gastric contents might be referred to hydrochloric acid production. With this in view I have attempted the comparative titration of about 30 gastric contents with methylene orange and tincture of cochineal, and in the investigation of 7 cases of typical "apepsia gastrica" with phenolphthalein, I found upon an average an increase of about 16 in the value of the total acidity in comparison with the values obtained with cochineal tincture. In 15 cases in which stagnation was present the differences were even greater; they amounted to between 18 and 21. In about half the cases these differences were compensated for by the values which I obtained with Leo's process, in the other half this difference was increased to a slight extent, and therefore we may say that among the conditions found in the stomach the cochineal tincture does not reveal acid phosphates. In my opinion, the influence of acid phosphates also deserves mention because of the values for total acidity which we obtain after administering a test breakfast and test-meal at the height of digestion, and because, as I was

able to demonstrate by special investigations, the acid phosphates of meat present in the evacuated contents at a test-meal may have caused an increase in the total acidity. I have permitted myself these brief remarks in order to show that some of the findings in gastric pathology which we have been accustomed to look upon as conclusive, in some points still admit of critical investigation.

Pepsin.—In definite cases of *extreme* subacidity, the quantitative estimation of *pepsin* may, under some circumstances, serve a practical purpose in diagnosis notwithstanding the fact, according to my own experience, that a *strict* parallelism between the production of pepsin and hydrochloric acid *does not exist.* Nevertheless—as I have seen in numerous investigations—with complete absence of hydrochloric acid secretion, appreciable quantities of pepsin are so rare—their entire absence is exceedingly rare—that in practice we will hardly fall into error if, in cases of high-graded subacidity in which the pepsin value of the gastric contents is but slightly above zero, we assume an "apepsia gastrica." From my own knowledge of the various methods employed to determine pepsin I advise only Mett's method, which for simplicity and exactness is preferable to all others, and which I myself used for more than three years. Even Hammerschlag's method, a modification of which I employed to increase the amount of albumin of the solution—using a Puro-solution in order to prepare a test-fluid—and the employment of specially chosen Esbach tubes which exactly equal one another, I have found neither more simple nor more exact. As the preparation of Mett's tubes as practised by me differs somewhat from the method given in the last publication on this subject, it is detailed somewhat more minutely in the footnote.[1]

Lab-Ferment.—Quantitative estimations of lab-ferment are very rarely necessary in practice, after making an examination for pepsin. Nevertheless, they serve a certain purpose (see later) and thus acquire some importance. Lab-ferment is either estimated according to the well known methods or, in the absence of an incubation oven—at least one suitable for

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[1] A part of the white of an egg is filtered through a piece of gauze into a small beaker glass or into a short, wide-mouthed test-tube, after which glass tubes about 2½ ccm. in length and about 2 mm. in breadth are slowly dipped into the filtered albumin. Air bubbles which rise in the glass tubes are permitted to disappear; this may be facilitated by slight tapping with the point of the finger upon the glass, which causes the air bubbles to rise. Then the beaker or test-tube with the tubes containing the albumin solution are placed in a perpendicular position in a large vessel of boiling water, and allowed to boil from five to ten minutes to coagulate the albumin. The flame is then removed and the glasses are permitted to cool slowly for a few hours. Now the test-tube is broken, or the coagulated albumin is cut out of the beaker glass, the tubes are freed from the albumin which adheres externally, and are preserved either in glycerin or in chloroform water. Prior to use the tubes are washed with water. After use the remainder of the albumin is removed, and the tubes may be filled as before with albumin.

the purpose—in the following manner: The patient is permitted to drink half a liter of milk, this is removed from the stomach one-half to three-quarters of an hour later, when the evacuated contents will show whether or not the milk is coagulated.

Steapsin.—The test for steapsin is up to the present of no practico-diagnostic importance; I can, however, absolutely confirm Volhard's results from my own researches. The test of the diluting secretion proposed by Roth and myself is as yet of no value in practice. The method I proposed for employing Gerber's acidbutyrometric test in the quantitative investigation of the diluting secretions and of resorption is only suitable for extreme grades of subacidity of the gastric contents. In regard to the method recently proposed by Sahli for testing the function of the stomach, v. Koziczkowski, under my direction, has shown sources of error, a fact which has, in the meantime, been confirmed by Lang and by Boeniger.

Other constituents of the gastric contents dependent upon the condition of the secretion have more of theoretic than practical interest, and, although not accurately developed in each individual case, are nevertheless worthy of brief mention. I have shown by means of systematic investigations that in high-graded subacidity the filtrate on the addition of a solution of iodin usually shows no change in color, or at most turns slightly to a yellowish-brown color, while in hypersecretion or hyperacidity the mass not rarely turns to a bluish-violet or blue color. Normal conditions are indicated by a Burgundy-red color which, in extreme cases, may have more of a violet, at times more of a brownish-red, tint. These peculiarities of the gastric contents have lately been confirmed by Bakman. The specific gravity, as I have shown in cases of subacidity, is usually increased—although not invariably—while in cases of hyperacidity and hypersecretion it as a rule—but by no means constantly—shows a decrease. According to my experience—and even with some reserve which is especially emphasized because exceptions occur—this is also true of the presence in the gastric contents of a dextro-rotary substance which may be determined with Pulfrich's refractometer called "refraction difference." I shall not enter here upon a discussion of the osmotic concentration of the gastric contents, although it has certain relations to secretion. In the meantime, I have also made numerous tests of the electric conduction of the gastric contents, and will only remark that these, as was to be expected, denoted a close relation to the amount of chlorin therein.

Passing from these preliminary remarks to the clinical consideration of disturbances of gastric secretion, we deal either with a decrease, that is, a loss, or an increase of secretion. In referring to *hydrochloric acid secretion* we speak of subacidity, that is anacidity (hypochlorhydria or achlorhydria), of hyperchlorhydria (hyperacidity [1]), and of hypersecretion

[1] From usage this term is applied especially to hyperaciditas anorganica.

(gastrosuccorrhea). Expressions such as hypochylia and hyperchylia, which are formed from achylia, as little convey the true meaning as does the word achylia, since chyle is entirely different from the gastric juice. The term heterochylia is quite misleading as it does not describe a substantive pathologic picture. On the other hand, it would be well to retain expressions like hypopepsia, apepsia and hyperpepsia for the reason that they clearly express the *effect* of gastric digestion without revealing more in regard to the nature of the secretory disturbance than terms which denote the state of hydrochloric acid secretion. This is self-evident when we reflect that the amount of hydrochloric acid contained in the gastric juice is the important and *decisive* factor for gastric digestion, because even a gastric juice rich in pepsin is incapable of producing a corresponding digestive activity without a large amount of hydrochloric acid. These designations are also suitable for the reason—as I have already remarked—that special disturbances of pepsin, lab-ferment, steapsin and the diluting secretion have as yet not been so fully recognized as to evoke any special therapeutic interest.

SUBACIDITY (APEPSIA GASTRICA)

In occupying ourselves first with cases of **hypochlorhydria** and **achlorhydria**, that is, **hypopepsia** and **apepsia**, we observe that they portray no comprehensive clinical picture, but, instead, a series of peculiarities common to the gastric contents. Among these the absence of free hydrochloric acid is the most important.[1] Other peculiarities, such as the coarse, fragmentary character of the evacuated gastric contents which, in cases of apepsia, reveals only traces of oral cavity digestion—not gastric digestion—showing low values for total acidity, usually a high specific gravity, high values for the dextro-rotary substance, the absence of staining products of amylolysis as well as the appearance of shreds of structure substance upon microscopic examination of the gastric contents, are as consequences of the decreased secretion merely of symptomatic importance, but may occasionally be of value in a clinico-diagnostic sense. Of pepsin and lab-ferment production we may say in general that they are more permanent than hydrochloric acid secretion, but, like the steapsin production in complete insufficiency of secretion, these may also be decidedly decreased or

[1] Naturally there are also cases with abnormally low total acidity in which free hydrochloric acid is still present, and also cases (rare) with normal total acidity and absence of free hydrochloric acid without lactic acid. As in the latter cases—they are usually associated with disturbances of motility in patients whose secretion is decreased to only a slight degree—the secretory energy of the stomach is insufficient for the production of the *physiologic plus* of free hydrochloric acid, these cases are looked upon as instances of "relative" subacidity, because every normal stomach at the height of digestion must show a physiologic plus of free hydrochloric acid.

entirely absent. According to Glaessner, only in carcinoma of the pylorus with complete or almost complete absence of pepsin may the distinct presence of lab-ferment be observed. That we miss a constantly recurring parallelism between acid values and pepsin is, in my opinion, explained from the fact that, besides other causes—according to the experimental investigations of Pawlow, Schiff, Herzen and others, there are various stimuli which promote in a one-sided way hydrochloric acid or pepsin production—in examining the total acidity we have considered too little the influence of phosphates, whose amount depends only partially upon the energy of secretion of the affected stomach. To this may be added that in former researches with the Hammerschlag process, the inaccuracy of the method has not been sufficiently taken into account in judging the results. In apepsia gastrica, at a time in which individual authors found different values for pepsin, I employed the Hammerschlag process with my own modifications, and in 4 instances I twice found the peptonizing ferment positively absent, and twice a property of peptonization of from 10 to 12 per cent., while on using the Mett process in 8 cases of apepsia gastrica, I found the property of peptonization only twice and then respectively 2 and 4 mm. In regard to the "diluting secretion," I proved in earlier investigations of the specific weight, and in more prolonged studies of the osmotic pressure of the gastric contents, as well as in quantitative estimations of the excretion of water by the gastric walls, that even in extreme secretory insufficiency, and especially in apepsia gastrica, it was still present, although apparently in diminished amounts.

In regard to the degree of total acidity in apepsia gastrica, I must reiterate what I have previously stated of the importance of phosphates in an estimation of the total acidity of the gastric contents. Although, as a rule, the majority of my cases of apepsia gastrica showed values for total acidity between 3 and 7—in a few cases I found the gastric juice neutral with phenolphthalein—I have, nevertheless, repeatedly observed cases with an acidity of 10 and 12, which possessed no property of peptonization, and at most showed only traces of combined hydrochloric acid (Leo's process). I have, therefore, made it a rule in suspicious cases of apepsia always to titrate with litmus and with tincture of cochineal.

As apepsia gastrica represents the highest grade of subacidity, so this affection distinctly demonstrates the clinical expressions of subacidity, its effects upon gastric and intestinal digestion, and upon metabolism; hence, in this description, I shall enumerate the causative factors and also the therapeutic indications which arise when a secretory insufficiency of the stomach is determined.

SYMPTOMS

The clinical signs which we observe in the higher grades of subacidity vary greatly. Often symptoms are entirely absent. At other times we

meet with indefinite complaints of sensations of pressure or fulness, or of eructation, partly of loss of appetite, partly of intestinal disturbance—particularly of diarrhea—or of more or less painful sensations in the abdomen. The great variety of clinical pictures in which we find subacidity as a symptom is not surprising when we consider that subacidity in a mild or severe degree accompanies or follows in the train of many maladies. Thus we find it not only in numerous local gastric affections among which carcinoma and alcoholic gastritis are probably the most frequent, but also not rarely in nervous and organic general affections, particularly in cardiac and renal disease, in pulmonary tuberculosis, and in the various disturbances of metabolism. For example, in the clinical history of 30 cases of persistent and "uncomplicated" apepsia gastrica which I observed for a long time, and of which I possess accurate notes, there were 8 cases of chronic alcoholism, 4 of pernicious anemia, 5 of advanced pulmonary tuberculosis, 3 of chronic nephritis, 1 of Graves' disease, 2 of well-developed chronic hysteria, 1 of trauma, 2 of severe erosion of the gastric mucous membrane (in both cases there were simultaneously symptoms of stenosis of the œsophagus leading to a "stenosed" form of gastritis). In 5 cases no especial etiology could be discovered. I have also observed transitory forms of apepsia gastrica in neurasthenics, above all, in a series of cases of delirium tremens. When I refer to an "uncomplicated" form of apepsia gastrica, in contrast to a "complicated" form, I have particularly in mind that prognostically ominous form which is an accompaniment of malignant neoplasm.

DIAGNOSIS

As it is especially important in the absence of a recognizable tumor and metastasis (if necessary, palpation per rectum and X-ray examination of the mediastinum)[1] to base the differential diagnosis on *the condition of the gastric contents,* I shall briefly include in this description the different diagnostic criteria, above all, the significance of an *admixture of blood with the stomach contents,* and the possibility of evacuating blood by lavage when the stomach is empty. In more than 6 cases which, by prolonged observation, were positively recognized as non-carcinomatous apepsia gastrica, I found blood in the stomach empty of food, and in some cases I detected shreds of tissue; therefore, I maintain that *only the combination of blood and pus indicates carcinoma.* As to the diagnostic inferences from the presence of shreds of tissue, I agree with Kuttner. In several cases in which I repeatedly found shreds of tissue upon lavage of the empty stomach, the patients complained of a gnawing sensation, sometimes of pain, and, upon taking food, of even more severe symptoms. I have con-

[1] Glandular tumors of the mediastinum have been actinographically determined by Weinberger (see his Atlas) and others.

sidered hemorrhagic erosions which, in common with Ewald and Boas, and in opposition to Einhorn, Parish, and others, I found in various gastric conditions, but I am not inclined to believe in the uniformity of a special clinical condition in hemorrhagic erosions. An increased vulnerability of the gastric mucous membrane I have quite often observed in cases of apepsia gastrica as a consequence of chronic nephritis, or as an accompaniment of chronic alcoholic intoxication. As apepsia gastrica may also be observed as a secondary accompaniment of true pernicious anemia, the differentiation of the forms of apepsia gastrica occurring in connection with carcinoma may, under some circumstances, be of practical significance. Here it must be borne in mind that pernicious anemia going hand in hand with apepsia gastrica, in contrast with carcinoma ventriculi, will, in spite of the severe anemia, usually present a well maintained panniculus adiposus, and that a thorough examination of the blood will generally reveal decided differences between these affections. For those cases of pernicious anemia in which there is no profusion of megaloblasts in the blood, I especially advise the minute observation of the leukocytes; for, in my numerous studies of pernicious anemia, I have found that with a diminution in the total number of leukocytes, which is quite usual, there is a relative increase of the mononuclear cells, while in the majority of cases of carcinoma, leukopenia is more rare, and the multinuclear cells are perceptible in normal or even increased amounts. Anemia without severe cachexia—it is true this is scarcely ever observed with the typical blood picture of pernicious anemia—may also occur in carcinoma of the lesser curvature which leaves both ostia of the stomach intact. In cases of this kind the nutrition of the patient is often but little disturbed, although hemorrhage from the ulcerating tumor may produce the picture of anemia (often to only a slight extent the picture of cachexia). In my experience cases of this kind are by no means so rare as we might suppose from the reports of various authors; for, among the cases of carcinoma which for several months I had opportunities of examining in the Clinic,—in the majority of cases, up to the time of death or of the operation,—more than one-third ran their course without symptoms of stenosis, and especially without the finding of lactic acid.

Practical interest—particularly therapeutic—is also elicited by those chronic intermittent diarrheas which, in my experience, are noted in about one-third of all cases of apepsia gastrica. We have long been familiar with these diarrheas, as well as with their relation to severe forms of subacidity, the condition having been lately described by Einhorn, Oppler, and others. In some instances they represent the most striking and unpleasant expression of the secretory disturbance, and they chiefly require observation for the reason that they may decidedly damage the nutrition. In my opinion, the tendency to diarrhea depends to a great extent upon the fact that the coarse ingesta, on which the stomach exerts no digestive

effect whatever, mechanically irritate the intestine, and also upon the fact that a larger number of bacteria with noxious influence upon the intestine have reached this organ than is the case with a normal secretion in which the virulence of one or the other of these bacteria has been attenuated. Moreover, the diarrhea may cause an injury to the general nutrition, since a disturbance of motility is associated with apepsia gastrica, and makes the ingestion of food insufficient because of vomiting, etc. The effect of a disturbance of motility combined with apepsia gastrica, that is, with a high degree of subacidity, becomes apparent when we examine the gastric contents from the fact that lactic acid may appear in such great amounts as to be easily recognized by the method I have indicated, with which, as is well known, we can only determine pathological amounts of lactic acid in the test breakfast or in the residue of the stomach before food is taken. That the absence of pepsin, as was first maintained by Hammerschlag, is necessary for the development of lactic acid, besides the combination of subacidity and motor insufficiency, I regard as not only unproven at the present time, but as most unlikely; for, under my direction, v. Aldor showed that in the artificial conditions of experiment, and under circumstances otherwise favorable for the production of lactic acid, pepsin by no means inhibits the appearance of lactic acid fermentation. Only lately I have had an opportunity of observing three cases of apepsia gastrica with temporary but undoubted lactic acid. Two of these cases occurred in perigastritis following cholelithiasis, and were confirmed by operation. The third case occurred in an elderly colleague who, since he was first examined two years ago, has continued in the best of health. I formerly made similar observations in a case of complete erosion of the stomach with the formation of numerous cicatrices and "stenosing pyloric hypertrophy," in a case of duodenal stenosis in consequence of tubercular peritonitis, as well as in a case of fat necrosis of the pancreas. Notwithstanding this, I am quite ready to admit that the finding of lactic acid in non-malignant conditions is so rare that, if continuously found for weeks, I consider an exploratory laparotomy to be not only justified but sometimes even absolutely necessary, provided we cannot certainly exclude carcinoma. In fact, my own observations of the frequent occurrence of lactic acid in carcinoma differ from the ordinary in that I succeeded in demonstrating its presence in only about 60 per cent. of my cases of gastric carcinoma, and I must particularly emphasize that among these cases were a large number in which an autopsy was conducted.

In regard to the cases in which an absolute cessation of hydrochloric acid secretion did not occur, the presence of combined hydrochloric acid, even without a quantitative estimation as I have previously remarked, may be assumed in all cases in which the total acidity, without lactic acid being present, reaches a certain amount; i. e., with the employment of phenolphthalein it exceeds 20, and, at the same time, the gastric contents reveal

positive signs that gastric digestion is present. If, after a test breakfast, the gastric contents without free hydrochloric acid show an acidity which exceeds 30, and if the examination for lactic acid, which should never be omitted in any case of subacidity, gives a negative result, we are probably right in assuming the presence of combined hydrochloric acid. As I have repeatedly observed in cases of stagnation combined with slight subacidity, in rare cases without the presence of lactic acid, higher values of total acidity—values from 50 to 60—may be found. I noted this particularly in cases in which ulcer of the pylorus gradually underwent carcinomatous degeneration, but also in non-carcinomatous cases of pyloric stenosis with a secretion which was not quite sufficient. In these cases, the gastric contents did not show the symptomatologic and simultaneous increase of the long bacilli which indicates lactic acid. Cases of this kind have not only confirmed my view that combined hydrochloric acid may inhibit lactic acid fermentation, but by their excellent chymification, as well as by their amount of erythrodextrin, they have proven that where large amounts of combined hydrochloric acid are present, notwithstanding the absence of free hydrochloric acid, we have the objective signs of proteolysis and of disturbed amylolysis. In regard to chymification, I must here remark that occasionally, even when large amounts of lactic acid were present, I succeeded in recognizing a slight degree of chymification in the gastric contents.

TREATMENT OF SUBACIDITY

Since it is my intention in describing the treatment of subacidity to consider the diminution of the secretory energy of the stomach only as a symptom of definite affections, I shall not fully discuss the treatment of these maladies, but shall limit myself in the main to the therapeutic indications necessary to combat this condition.

Diet.—Beginning with the dietetic treatment, we find, fortunately, entire unanimity between theory and practice. This is first shown by the requirement that the food be finely divided, and of such a character that it will pass the pylorus as under normal circumstances. The importance of this therapeutic principle, so long established in practice, we can appreciate after we understand that the reduction of bread and pastry to a fine, pappy mass is not only the function of mastication in the oral cavity, but also that of proteolysis in the stomach. As I have demonstrated by microscopic investigations, a small portion of bread (consisting of gluten albumin) may be compared to tissue or a sponge in whose meshes the starch granules are deposited. To secure this reduction of food with defective proteolysis, good teeth are absolutely necessary, therefore in many cases of extreme subacidity the examination of the teeth by a dentist and the correction of defects form the first part of the treatment. The thorough cleansing of the oral cavity is also necessary, for the reason that the stomach of the patient in question can offer but slight resistance to the bacteria

descending from the mouth. In patients suffering from subacidity thorough mastication is also necessary for another reason. Recent investigations (Pawlow, Schüle, Troller, Riegel and Schreuer, as well as the experiments conducted by Martin Cohn under my direction) have shown that, in cases in which the property of secretion has not been entirely lost but is capable of increase upon decided stimulation, thorough chewing will, in fact, increase the production of acid. Therefore, slow eating and thorough mastication are to be especially insisted upon with those patients who suffer from subacidity. As a rule, crisp bread and pastry which are well ground up by the teeth, such as crackers, zwieback, toast, etc., are the best. The more tender varieties of meat are given, such as poultry, veal, etc., in place of that containing coarse fibers held together by a tough connective tissue network (beef, lamb), and we should see that the food is so prepared in the kitchen as to facilitate its mastication. According to the investigations of A. Schmidt, in which I entirely concur, too thorough cooking of meat, that is, broiling which hardens the connective tissue which is not subsequently dissolved in the intestine in apepsia gastrica, may irritate the intestinal wall and thus produce diarrhea. Raw meat should not be given to the patient suffering from subacidity. Gelatinous foods (gelatin) are very useful since gelatin dissolves into fluid in the stomach. In those cases in which it is impossible to administer sufficient nourishment in the manner described, as well as by the contingent use of artificial foods, the employment of means for disintegrating food, for example, Collin's apparatus, or masticators, or small meat choppers, are advisable, and it is well for the patient to use the apparatus himself since food brought upon the table in a broken-up condition is less stimulating to the appetite than that served in its original form and then finely divided by the patient himself. This point, in my opinion, is all the more worthy of consideration because the preparation of food and its appetizing appearance are important matters to a patient suffering from subacidity. Pawlow has shown that the appearance of food has a stimulating or depressing effect upon the appetite which materially influences the intensity of the secretion of gastric juice. We may truly say that the sight of appetizing food not only makes the mouth water, but also causes the gastric juice to flow, and to the joy of every gourmand, we maintain that food eaten with zest is half digested. For this reason, in all cases of subacidity in which the glandular parenchyma still reacts to stimulation by a secretion of gastric juice, great weight must be attached to the attractive and appetizing preparation of the food, which is obvious from the fact that many of these patients already suffer from loss of appetite. Therefore spices, meat sauces, and the other condiments of the kitchen—within certain limits—are just as necessary here as the stomachics of the apothecary. In regard to the latter, I must mention in passing that they act upon the nerves of smell and taste and thus produce a feeling of desire which reflexly influences gastric secretion. It is evi-

dent from the foregoing that not only bouillon but even the so-called appetizers should be advised as overtures to a meal in all cases of this kind. When the auxiliary measures here mentioned do not sufficiently break up the ingesta, and also stimulate the gastric secretion, we must at the onset dissolve or very finely pulverize the greater part of the food. As to albumin, milk and eggs are to be utilized in different ways prior to or simultaneously with artificial foods. In the use of milk, it is true that great care is necessary in patients who show a tendency to diarrhea, and the advice of A. Schmidt to administer to such patients only the salicylated milk—particularly in summer—is especially valuable. In cases in which there is a tendency to diarrhea I have repeatedly seen favorable results from well-sterilized diabetic milk (almost free from sugar), which I used chiefly in cases of diarrhea. Of artificial food preparations—thanks to the zeal of busy manufacturers—a large number are at our disposal, so that in the choice in a given case we pay more attention to the taste, or, in other cases, to the price of the article. These food preparations are best given in soups, in milk, or in purée. The various meals should not be large, and we should rather adhere to the law, "Little and frequently" than to "Much and rarely." Of fats I prefer the easily digested milk fats (milk, cream, butter), whose secretory, inhibitive influence in severe forms of secretory insufficiency is no longer of much importance.

Physical Treatment.—Physical treatment generally aids us but little in controlling subacidity. In so far as my own experience and investigations permit an opinion, neither hydrotherapeutic measures, nor massage, nor electricity of the stomach can claim a positive influence upon the secretion of gastric juice. *Gastric lavage* may also be tried in subacidity when there are large quantities of mucus in the empty stomach, although in the majority of such cases the use of alkaline mineral waters upon an empty stomach is sufficient (washing in the direction of the pylorus), and also when a disturbance in motility is combined with subacidity. In regard to the balneotherapeutic treatment of subacidity, I prefer the waters containing sodium chlorid (Kissingen, Homburg, Wiesbaden, etc.), but experiments to demonstrate the relation in such spa cures of the sodium chlorid in the organism to the secretion of hydrochloric acid lack a satisfactory scientific basis. The secretion of hydrochloric acid can nowise be increased by the rectal introduction of sodium chlorid, as I have proven in a number of patients suffering from subacidity. This is the result I expected inasmuch as, in the patients who here come into question, the secretory insufficiency is to be attributed more to the pathological condition of the secreting cells and the nerves which stimulate them than to a deficiency of sodium chlorid in the organism. In cases in which a general neurosis is the cause of subacidity, the elements of physical treatment may, under certain circumstances, indirectly act favorably upon the production of hydrochloric acid.

Drug Treatment.—Drug treatment of subacidity represents on the one hand a substitution therapy, and on the other hand an attempt to increase the diminished secretion. Substitution therapy aims to supply hydrochloric acid and the ferments which have been lost—particularly pepsin—from without, and probably represents, so far as the introduction of pepsin is concerned, the oldest form of organotherapy upon an exact basis in man. If we study the consequences of subacidity from the introduction of hydrochloric acid and of pepsin, a critical judge will not place a high estimate upon the effect of such treatment; for, as the investigations of Pfungen, Schüle and others have demonstrated, the amount of hydrochloric acid combined by the proteids compared with the quantity of hydrochloric acid therapeutically administered by the mouth is so great that the introduced hydrochloric acid can hardly be looked upon as a substitute for the secretion which has been lost. Moreover, in regard to pepsin it must be stated that, with adequate quantities of hydrochloric acid, even slight amounts of pepsin are enough to produce a satisfactory proteolytic effect; that, however, the introduction of pepsin is useless if, during the entire duration of digestion, a sufficient quantity of hydrochloric acid is simultaneously present in the stomach. Benefit from the various wines of pepsin, therefore, is probably because of the stomachics they contain. In cases with complete loss of secretion it is far wiser to use the pancreas preparations which act upon a neutral or alkaline floor and permit digestion of the small intestine to begin in the stomach; that is, where gastric digestion is lacking, intestinal digestion is aided by the administration of proper preparations. Among these preparations *pankreon* has been most serviceable in my hands, while from papain, which is said to be active in an acid media, I have seen no remarkable results nor have Grote, Hirsch and others. In regard to a special form of substitution therapy, the administration of canine gastric juice, which was first advised by Pawlow and subsequently by Russian and French investigators—in Germany this has only been employed by Paul Meyer—I have no personal experience. I do not believe, however, that this method is often absolutely necessary, nor, the material being so difficult to obtain, that it will come into general use.

Great value was formerly attached to certain products that increased secretion, and Pawlow by masterly investigation and technic has now made it possible for us accurately to study this question experimentally. Pawlow found that raw meat, meat juice, meat broth, Liebig's meat extract, milk, gelatin, certain peptones and large quantities of water stimulate the gastric juice. Herzen compared dextrin (not chemically pure) and Liebig's meat extract, and found that the combination of large doses of these (25 to 50 grams) in a dog with gastric fistula stimulated the gastric secretion and formed pepsin. Employed alone, dextrin particularly influenced pepsin formation, while Liebig's meat extract chiefly acted upon the gastric juice. Alcohol proved a mighty stimulant to the gastric secretion but had abso-

lutely no effect upon the production of pepsin. Alcohol administered per rectum also showed itself a stimulant to the gastric juice, as Metzger determined in Riegel's Clinic. Herzen reports that he produced favorable results in man with a mixture of Liebig's meat extract and dextrin in a palatable form. Another preparation, inulin, after the administration of which Herzen found a marked amount of pepsin in the gastric juice if he simultaneously administered alcohol, I have employed in a few cases in the form of a tea containing inulin (25 grams in 300 c.c. of the tea for a trial breakfast). Although I have in a few cases seen a slightly stimulating effect upon the gastric juice, yet, considering the expense of inulin, this was not so marked as to tempt me to make very extensive experiments.

It may be urged in opposition to the explanations given here that there are cases of apepsia gastrica which run their course for years and even decades without any marked disturbance, and we may conclude from this that it is not always necessary in cases of subacidity to follow the outlined treatment here suggested. In reply it may be maintained that no proof has yet been furnished that the persistent functional over-exertion of those glands of the intestine which furnish the proteolytic ferments and their combinations will never produce injurious effects. What we have stated of the relation of certain forms of diarrhea to apepsia gastrica, and the favorable influence upon them of measures here described, by no means strengthens the arguments of those who declare these measures to be unnecessary; and even if we could adduce no positive facts in support of the fundamental principles here developed, the purely theoretical assumption that prophylaxis is the best therapy is sufficient reason and justification. This is particularly true of the suggestion of small and frequent meals, since these, more than any other means, conserve the motility of the stomach, the weakening of which at once disturbs the equilibrium of the patient's metabolism. A disturbance of motility in cases of apepsia gastrica soon changes the stomach from a natural disinfectant apparatus into an incubation apparatus for various microörganisms—under some circumstances, also the pathogenic —and, from a teleologic point of view, a reason may be discerned in the facts that the stomach in apepsia gastrica usually empties itself more rapidly than under normal circumstances, that it is unable to digest its contents, and also that it has no power upon micro-parasites, but rapidly propels them into the small intestine, in which a great number of microörganisms appear to perish.

HYPERACIDITY

In the description of an *immoderate* secretion of acid we must sharply differentiate those conditions in which gastric juice abnormally rich in hydrochloric acid is excreted *only by the stimulation of the ingesta* from those in which gastric juice is excreted with a *permanent secretion—one independent of the introduction of food.* We must, therefore, sharply dis-

tinguish hyperacidity (superacidity, peracidity) from hypersecretion (supersecretion, parasecretion, gastrosuccorrhea). As in the former the stomach reacts only to the stimulation of the ingesta present, and produces a secretion powerful in digestion, in the second case a secretion takes place without stimulation, and, as to cause and effect, the stomach in the former case bears the same relation to the latter as a pump or open well to an artesian well. But since it is not my purpose here to discuss the question in how far hyperacidity is a substantive disease or is merely a symptom of other affections, I shall at once define our chemical conception of hyperacidity. To express this in figures is very difficult, for, on the one hand, the amount of secretion varies greatly under physiological conditions in one and the same individual as well as in different persons, and, on the other hand, the sensibility of individual persons to an excess of hydrochloric acid in the stomach shows extraordinary variations. In practice, this latter factor merits special consideration because only that patient suffering from hyperacidity seeks a physician who suffers discomfort on account of his disturbance in secretion. In my opinion, therefore, a description of hyperacidity necessitates the simultaneous delineation of all those factors which produce a more or less marked acid hyperesthesia. Besides, our conception of the condition, that is, whether we base the chemical conception of hyperacidity on the total acidity, or make free hydrochloric acid most prominent, varies greatly. Among those who adhere to the first theory, Ewald estimates hyperacidity in a total acidity between 60 and 70, and Rosenheim above 60; Johnson and Behm consider a gastric juice with a total acidity of 70 as hyperacid, and Boas regards in the same light a gastric juice with an acidity of over 2 per thousand. Riegel, besides laying much stress upon the total acidity, also attaches great significance to the proportion of free hydrochloric acid. "The most important point is the free hydrochloric acid. Only where this is increased are we justified in speaking of an actual hyperaciditas hydrochlorica. Values of 60, 70, 80 and more after a test-meal, of 50 to 60 and upwards after a test breakfast are frequently found here." Schüle considers the gastric juice to be hyperacid when it reaches a height of 0.22 per cent. more than free hydrochloric acid, and shows a total acidity of more than 70. Schneider maintains that a gastric juice with more than 0.25 per cent. of free hydrochloric acid is to be looked upon as hyperacid, and that we may also regard as hyperacid a gastric juice which still gives a reaction for free hydrochloric acid with a dilution of more than ten times its bulk. Although it must be admitted without more ado that gastric contents which with a total acidity of 70 give an intense reaction for HCl may be called hyperacid, yet, in doubtful cases I should be reluctant to diagnosticate hyperacidity of the gastric juice upon this single symptom of the gastric contents, but only upon the sum of peculiarities which the test breakfast or the test-meal enable us to recognize. For we may examine gastric juice in which titration shows no ab-

normally high degree of total acidity, while, notwithstanding this, certain peculiarities of the gastric contents and also the clinical symptoms of the patient may correspond to the condition which we are accustomed to find in hyperacidity. Cases of this kind which Schüle under my direction described by the name of hyperaciditas larvata or hyperaciditas occulta are not extremely rare and, in a certain sense, are the antitheses of those cases of hyperacidity running their course without symptoms in which the examination of the gastric contents reveals the distinct characteristics of hyperacidity while the patient presents absolutely no symptoms.

The typical peculiarities of the gastric contents in hyperacidity are the following:—

The thin fluidity of the gastric contents, the tendency rapidly to form two layers, an upper one consisting of fluid and a lower one of a finely pulverized, starchy sediment, and, especially prominent, its conspicuous chymification. In regard to the layers, we find that if the gastric juice is placed in a graduated test tube for two hours after its withdrawal, and spontaneous sedimentation is permitted, the "layer quotient," i. e., the figure which the height of the sediment shows divided by the total amount of gastric contents collected for sedimentation, is frequently—although not invariably—below 50 per cent. Sedimentation—contrasted with that of many subacid gastric contents—not only occurs very rapidly but most completely. Besides amylorrhexis of the hyperacid gastric contents, amylolysis also merits some consideration. As I showed years ago in a large number of cases of hyperacidity, relatively low values for the dextro-rotary substance were found in the majority, and (particularly dependent upon this) also a low specific gravity of the gastric contents,[1] as well as frequently a Bordeaux red or violet—in rare cases also a blue—reaction of the iodin test. In some cases I detected an abnormally low molecular concentration of the gastric contents. As to the proportion of free hydrochloric acid to total acidity, in a comprehensive examination of my cases of hyperacidity in which the total acidity amounted to more than 60, more frequently than otherwise I found the free hydrochloric acid equalled two-thirds and more of the total acidity. In nine cases of hyperaciditas larvata in which the total acidity did not exceed 50, I only twice saw the acidity value of free hydrochloric acid fall below 50 per cent. of the total acidity, while in other cases in which the total acidity was not over 50 the value for free hydrochloric acid was more often below than above 50 per cent. of the total acidity. These conditions, however, are not so characteristic in the individual case as to be of direct diagnostic use. The relation of the total amount of the gastric contents here calls for consideration, and all the more so because in various places erroneous views have been promulgated

[1] I am unaware that either Schüle or I have given a specific gravity of over 1.020 as a "positive" sign of subacidity, as has recently been alleged by Illoway, nor can this statement be proven.

as to the influence of motility in hyperacidity. For a long time it was remarked that one hour after administering the test-breakfast in cases of hyperacidity, and upon the evacuation of the gastric contents, an increased amount was not rarely evacuated. This fact was long known to those who only recently relinquished the antiquated method of estimating the total amount of gastric contents solely by expression, that is, by aspiration, *without subsequent lavage. For more than eight years, after every investigation of the gastric contents* I have made an exact estimation of the residue of the gastric contents by the aid of a mathematical formula which is based on the relation of the specific gravity of the undiluted gastric juice to that of the gastric juice diluted with 100 c.c. of water, this principle, as I learned subsequently, having been previously employed by Jaworski in his scientific investigations. I still employ this mode of calculation because the estimation of acidity according to the method of Mathieu and Rémond in cases of anacidity leaves us in the lurch; that is, only by the employment of a complicated modification given by Cohnheim are we enabled to reach a conclusion. It may be interesting to note here that the amount of gastric contents I have obtained in a great number of investigations in normal persons one hour after a test breakfast reached on an average the figure of 150 c.c., while in 40 cases of hyperacidity I obtained an average figure of 210 c.c. (the lowest being 124 c.c., and the highest 400 c.c.). Contrary to numerous authors, I do not believe this increase in the amount of gastric contents one hour after the administration of the test breakfast—at least in uncomplicated cases—to be the consequence of a so-called "atony" (an expression which had best be eliminated from the nomenclature of gastric pathology since it is not only superfluous but, in consequence of the dissimilar significance attached to it by different authors, is liable to cause confusion) but to be the product of an increase in the secretion of the gastric juice of a hyperacid stomach. In numerous researches in uncomplicated cases of hyperacidity I was unable to detect any sign of motor insufficiency either by the currant test, by the fermentation test, or by microscopic examination of the evacuated gastric contents. Perhaps the fact, which has already been alluded to, that the "layer test" of hyperacidity usually gives a lower layer quotient than in subacidity, and that occasionally a conspicuously low value for the molecular concentration of the gastric contents is found ("hydrorrhœa gastrica") may be utilized in the same sense as my explanation. The view that the increase of the gastric contents in hyperacidity is to a great extent the consequence of an increased secretion is somewhat favored by the experience that, in contrast to the condition, just described, of the test breakfast (rich in starch), upon utilizing the test-meal relatively rich in albumin and poor in carbohydrates, an extremely rapid emptying of the stomach may frequently be observed; this phenomenon may be satisfactorily explained by the theory that the test breakfast, by the too early interruption of amylolysis ("Secretio celer et

alta "), and the disturbed liquefaction of the introduced starches, promotes an increase of secretory stimulation. Johannes Müller admits that the digestion of bread is generally delayed by a decidedly acid secretion, and that the inhibition of amylolysis appears to be influenced more by the rapidity than by the intensity of the acid secretion.

SYMPTOMS

As already indicated, the clinical manifestations of hyperacidity vary in individual cases to an extraordinary degree. One case will run its course without symptoms, and another with excessive pain which occasionally reaches such a degree and is so frequent that signs of chronic under-nutrition and loss of strength may appear. Complications such as erosions, fissures with consecutive pylorospasm, ulcer of the stomach, and especially ulcer of the pylorus, may in isolated instances impress their stamp upon the case. However, except for such "complications," in the great majority of cases of hyperacidity we may construct a clinical picture which, although not applicable to all cases, nevertheless applies in a great many. The patients are usually thin—but not always—are, as a rule, between 20 and 40 years of age, the majority of them belong to the erethismic type, they complain of gnawing, painful sensations of pressure in the gastric region, and state that these unpleasant symptoms occur as a rule several hours after eating—after the midday meal, or in the late afternoon hours—they continue for a time and then disappear. The mechanical state of the food is said to be unimportant, and the pains to disappear frequently by the ingestion of milk or by a large dose of sodium bicarbonate. The appetite is good, but the patients refrain from eating through fear of pain (cibophobia). Vomiting is rare, but eructations are frequent, as well as nausea and retching. The patients usually complain of chronic constipation. Provided complications or other diseases which, according to experience, play a rôle in the etiology of hyperacidity (pylorie stenosis, gastroptosis, hernia epigastrica, etc.) are not present, objective investigation in most cases reveals only slight diffuse sensitiveness on pressure. Frequently even this is absent.

Hyperacidity is observed in different countries in varying frequency. According to Jaworski it occurs in Lemberg in 51.8 per cent., according to Einhorn in New York in 51 per cent., according to Johnson and Behm in Stockholm in 36.4 per cent., according to Kövesi in Budapest in 30.4 per cent., according to Mathieu and Rémond in Paris in 29 per cent., according to Bouveret in Lyons in 25 per cent. of all cases of gastric disease which come under observation. In my own experience it is noted in Berlin in scarcely more than one-third of all gastric cases. At all events, among the clinical cases received at the Charité in Berlin, the disease is decidedly less frequent than it was in the Clinic in Giessen, as I had an opportunity of observing.

ETIOLOGY

The causes of hyperaeidity also vary in the individual eases, and are partly of a loeal, partly of a general, nature. Among the local factors ehemie, thermal and mechanieal irritants play a rôle which, upon prolonged aetion, may perhaps also lead to parenehymatous ehanges in the gastric mueous membrane. I believe it very likely that the eonstant use of food rieh in spiees and the generous consumption of aleohol—especially of sour Rhine wine—play a rôle in the etiology of hyperaeidity, and I also take eognizance of the thermie faetors because hyperacidity is relatively eommon among bakers, who, as is well known, frequently eat hot pastry. Moreover it is by no means eertain that the frequent drinking of ice eold fluids upon an empty stomaeh does not predispose to hyperacidity. In addition there is a large group of loeal and general neuroses—essential, toxie (tobaeeo!), or arising reflexly [1]—and, what I believe to be worthy of serious diseussion, the ehronie eonstipation so often noted in hyperacidity. With their removal the symptoms of hyperacidity frequently disappear. By some authors (Hemmeter) eontinuous over-stimulation with a meat diet is eonsidered of speeial etiologie importanee, and Westphalen even discriminates a eongenital predisposition attributable to the predominant meat diet of preeeding generations. Cloetta has also shown that in dogs of the same litter, those nourished with meat show free hydrochlorie acid after a meal while in dogs fed with milk it is absent. Although Cloetta eould find no absolute differenee in the histologie strueture of the mucous membrane of these dogs on different foods, I believe it at least possible that organic ehanges may be produeed in the gastrie mucous membrane by hyperaeidity. There is, as I have proven in several of my eases, a true *acid gastritis,* i. e., a eombination on the part of the stomach of the produetion of mueus with that of hyperacidity. Sueh cases upon lavage of the empty stomaeh not only show more mucus than in the norm—the lavage water of a normal stomaeh before food is taken is grey, has a turbid appearanee, and resembles the water after eleansing the mouth—but the microseopie examination of the peeuliar mueus plugs from the test-breakfast also shows a eonspieuous number of glistening nuelei of leukocytes with completely digested protoplasm. The eonclusion is obvious that the inerease of aeid produetion in sueh eases is not only funetional but, perhaps, also anatomical, espeeially in the eases of hyperaeidity running their course with motor insuffieiency, and in whieh the autopsy reveals proliferation of the glandular parenehyma.

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[1] Some cases of hyperacidity and gastroptosis, hernia epigastrica, and local peritonitis may also be of more or less neurogenic origin.

TREATMENT OF HYPERACIDITY

In the treatment of hyperacidity diet occupies the first place. Its importance is evident from the fact that, probably, in no branch of the treatment of gastric affections are the fundamental laws of nutrition so warmly disputed as in this instance. The question chiefly resolves itself into this, whether preference should be given to albumin which readily combines acids, or to starches which are less stimulating to the production of hydrochloric acid in the patient suffering from hyperacidity. To-day, when the waves of discord have become somewhat smoother, we may say that occasionally in this discussion the point of view has been too one-sided, inasmuch as it has been overlooked that in the dietetic treatment of the patient the entire organism is to be considered as well as the stomach, and that in the consideration of dietetic experiments and experiences, meat and albumin have often been regarded as synonymous with carbohydrates and vegetables. In some of the earlier researches, too, the importance of the employment of "equicaloric" amounts of ingested food, and differences in the composition of the food, have not been sufficiently taken into account in the individual experiment, as is apparent from the special preparation of the food, its volume, and the labor necessary in masticating it. Finally, in my opinion, the varying etiology of hyperacidity from the basis of nutrition has been too little considered in the individual cases. A certain bias of observation has until recently been manifest, for, in the question under discussion the cry has almost always been "here albumin" or "here carbohydrates" while of fat we have only been told that patients suffering from hyperacidity "may" have good butter, or that large quantities of fat should be avoided for the reason that they readily disturb intestinal digestion by the hyperacidity of the gastric juice. Fat, at the present time, not only occupies a position of equal importance with the two other nutritive products in the diet of the person suffering from hyperacidity but even a preferred position, for the reason—as we know from the pioneer experiments of Pawlow upon the dog, and those which I simultaneously carried out with Akimow Percz, and the subsequent ones of Backman and others in man—that fat inhibits gastric juice secretion, and therefore is utilized normally in the intestinal canal of the patient with hyperacidity, yet does not inhibit the motility any more than an equal quantity of other caloric food products. The *curative* power of large quantities of *milk fat* in the dietary of hyperacidity thus appears to be proven, accordingly fat, especially milk fat, has been accorded a permanent place in the treatment of hyperacidity. In earlier investigations which I made regarding the relation of the dextrorotary substance in the human gastric contents, I found in concentrated solutions of sugar another dietetic measure to diminish gastric juice secretion. Similar researches have recently been made by v. Aldor in man, and by Clemm in the dog, with gastric fistula, and the results have been con-

firmed. For the removal of the acid symptoms, concentrated sugar solutions are most effective if administered to the patient only upon their appearance. Instead of sugar solutions we may use honey, although, as Clemm has shown, levulose lacks the powerful, inhibiting secretory effect of dextrose. For the rational treatment of hyperacidity, however, dietetic measures to diminish gastric juice secretion are by no means sufficient, but we must simultaneously remove from the diet all those articles which in themselves possess the property of increasing the secretion of the gastric juice. As food and dainties in this respect have already been discussed in the description of subacidity, I desire only to urge the utmost care in the free use of extracts of meat (concentrated bouillon, sauces), and the spicy preparation of food in general, above all, such food products as require prolonged mastication. A rational *arrangement of the meals* also appears to be highly important in hyperacidity. It is directly opposed to the true conception of the nature of hyperacidity that a patient with this affection should take frequent but small meals. The essential feature of hyperacidity consists in an abnormal irritability of the secretory apparatus of the stomach, the expression of which is not manifest during the period when the stomach rests, but only during its time of labor. For this reason I maintain, with A. Schmidt, that but a few and—provided there are no definite factors to the contrary—somewhat larger meals are indicated.

In regard to the *mixture* of individual food products, I agree with those who have lately recommended a vegetable or a lacto-vegetable *régime* in the treatment of all those cases of hyperacidity in which this appears to be the consequence of a functional neurosis, or in which the symptoms of the latter at least dominate the clinical picture. According to my experience, in the remaining cases, the administration of a mixed diet is best,—one which, in a normal calory amount, contains considerable fat (from 150 to 180 grams)—especially milk fat, such as cream, butter, Jaworski's "kraftmilch," or Gaertner's fat milk, fat cheese, oil (according to Walko, even by the stomach-tube), emulsions of almond oil, etc., but not bacon and beef fat—as well as a normal quantity of albumin (amounting to about 120 grams) only a small part of which should consist of meat, preferably broiled meat, or of fish. Starches had best be administered in the form of thick soups, gruels (mashed potatoes, flour, rice, etc.), and tender, well-cooked vegetables dressed with considerable butter; it is advisable in general not to give more than 250 or 300 grams, but the amount permissible depends very much upon the form in which it is given. As soups rich in extracts are usually unsuitable for patients with hyperacidity, carbohydrates, if not fluid or also taken in other ways, may be given in flour soup, milk soup or fruit soup. Bread, whenever possible, should be rich in albumin (aleuronat, roborat or casein breads) or fat containing, as bread and butter or Rademann's nutritive toast (Nährtoast), etc. Sometimes foods containing oil (sardines in oil) may be permissible, or a dose of

emulsion of almond oil before meals, or a not too strong fatty cheese, as, for example, the crême double de Normandie, which gourmands even eat with sugar, or with whipped cream and sugar. The effect of the latter, as has already been stated, justifies the use of sweets, such as fruit jellies, creams, sweet stewed fruits, and fruit syrups, such as raspberry syrup, grape juice, etc.—also in the form of Nectar and Pomril—as well as malt extract, malt beer, porter, and honey; in this respect the particular idiosyncrasy of the patient suffering from hyperacidity is to be considered. Among wines, as a rule, red wine is preferable, which had best be diluted with an alkaline mineral water. Among the Rhine wines, the acid varieties must be avoided; white Bordeaux wine and white Burgundy are preferable to Moselle wine. The tolerance of beer varies greatly in the individual case; in general, those rich in carbohydrates are preferable to those deficient. Coffee is usually badly borne; tea and cocoa frequently agree well with the patient, particularly if given with large quantities of cream.

Physical Treatment.—The curative factors of physical therapy from a symptomatic standpoint may aid us greatly; moist, warm compresses as well as dry heat (thermophore, Japanese stove) are particularly effective for the unpleasant sensations produced by hyperacidity. Simon maintains the favorable influence of sweating upon the gastric juice secretion, which coincides with the researches Edel made in Riegel's Clinic. I have been unable to find these so frequent as to make the method worthy of general acceptance. However, other hydrotherapeutic measures are not rarely indicated in those cases going hand in hand with neurasthenia or attributable to this.

Drug Treatment.—Drug treatment is employed in hyperacidity for various purposes. For the hyperacidity itself subcutaneous injections of atropin were advised by Riegel as valuable on account of the experiments which he made in dogs with fistula, and from his own clinical experience. Unfortunately, however, the dose required decidedly to inhibit secretion is so great that atropin can only be employed for relatively brief periods daily, unless we wish to run the risk of poisoning from atropin. For this reason atropin in doses of one milligram is particularly indicated in the treatment of acute exacerbations of hyperacidity. Whether bismuth and silver nitrate also possess the power to inhibit secretion is not certain. In systematic investigations of the gastric juice secretion prior to and after treatment with bismuth and silver nitrate lasting for several weeks, I have personally been unable to determine any absolute decrease of the gastric juice secretion. Nevertheless, these drugs at least deserve mention.

Alkalies for a long time quite properly played a rôle in the symptomatic drug treatment. It is wise to give the alkali or the alkaline mixture—usually a mixture of various alkalies is given to which, in cases complicated with constipation, a laxative salt is, as a rule, added—to the patient only on special indications, i. e., only when acidity becomes noticeable, and

then not in small, divided doses but at once in a decided dose (at least what will cover the tip of a knife, and, if necessary, from one-half to one teaspoonful). The immediate effect of the alkaline treatment upon the symptoms in hyperacidity proves more absolutely than any theoretic consideration that a causal relation must exist between the excess of acid and the manifestation of the symptoms. It appears that by the administration of alkalies this plus of hydrochloric acid which has a disagreeable effect upon the patient is neutralized. The prompt action of alkalies may very well be explained by the assumption that a pyloric spasm and its consequences (painful contraction, retention of gases, etc.) are arrested by neutralizing the acid, or that in hyperesthesia gastrica the acid irritation is removed. That the alkaline therapy is injurious is neither maintained by those who have had great experience in this treatment of hyperacidity, nor by other observations such as have been made, for example, in the treatment of acidosis with large doses of an alkali. An excess of alkali is removed from the body with relative rapidity. In numerous original experiments, on the administration of 10 grams of sodium bicarbonate, alkalinity of the urine usually occurred in from ten minutes to an hour and a half, and rarely lasted longer than sixteen hours. The administration of alkalies to patients with hyperacidity may, under certain circumstances, even increase amylolysis, which in practice is all the more striking since in the treatment of hyperacidity the resistance of saccharifying ferments accomplishes but little. In association with Stargard I determined this for the remedy last advised, takadiastase.

The treatment of hyperacidity naturally remains incomplete so long as only the irritative condition of the gastric mucous membrane is combated, and not the *cause* which produces the condition. Although this is not the place in which to consider this subject minutely, nevertheless it must be here emphasized, above all, that in a great number of cases of hyperacidity the simultaneous *constipation* largely demands our therapeutic interest, for, in fact, it almost gives us the impression that hyperacidity may often be more readily cured by attacking the intestine than the stomach. In a certain sense the relations are here inverted, as in the diarrhea of apepsia gastrica. Therefore, at least in all cases of hyperacidity in which the bowel action is not perfectly normal, a systematic Carlsbad cure—either with natural Carlsbad water or Carlsbad salts—is indicated, and all the more so as in cases of hyperacidity without constipation we occasionally observe very decided improvement under the influence of Carlsbad salt or Carlsbad water. In every individual case of hyperacidity we should, however, as therapeutists, reflect upon the limits of our differential diagnosis, which in this affection are not infrequently revealed when a case which we have believed to be "only" hyperacidity proves to be an ulcer of the stomach, and demands of us special consideration in the treatment.

HYPERSECRETION

In contrast to hyperacidity, hypersecretion (parasecretion, gastrosuccorrhea, Reichmann's disease, etc.) is distinguished by a continuous "flow of gastric juice," therefore is a condition in which the stomach, even *without the stimulus of the ingesta,* secretes gastric juice. Although hypersecretion is a substantive condition, nevertheless it is not an etiologic unity, for it arises under varying circumstances. Its recognition depends upon the proof of greater or less amounts of secretion in the stomach devoid of food, and we will therefore first consider the question, what conditions, under normal circumstances, promote secretion in the empty stomach? As investigations of this point by various authors—I shall mention here Riegel, Rosin, Martius, Schreiber, Gintl and others—have furnished results in part contradictory, in answering this question I shall primarily relate my own observations. Years ago, in experiments which I made in Ewald's wards of the Augusta Hospital in Berlin, I found in 38 persons that noteworthy quantities of actual gastric juice were not present in the normal stomach, but I noted the same condition in several hundred observations which I had an opportunity of making in the course of years while introducing the stomach-tube into the stomach empty of food—partly for diagnostic, partly for therapeutic, and partly for clinico-experimental purposes. Therefore, I agree with Riegel, Ewald, and numerous other authors even more fully to-day than formerly in believing that the presence of appreciable quantities of fluid which presents all the peculiarities of gastric juice is abnormal, and that in cases in which this fluid reaches a certain amount, say exceeding 30 c.c., we have a pathologic condition.

What are the properties which lend to a fluid the *characteristics of the gastric juice?*

The fluid which usually flows in a stream from the tube is, as a rule, clear as water or turbid, of a light grey color, or may also show a greenish discoloration. The latter change is readily produced in the fluid from the fact that during the act of expression the pylorus occasionally opens and bile regurgitates from the small intestine into the stomach. In numerous observations which I had an opportunity of making, the closure of the pylorus during the act of expression is more readily overcome when fluid is present in the stomach than when it contains a thick, pappy mass. The pure secretion of the empty stomach, compared with the "residue" which represents remains of food containing chyme without a distinctly recognizable admixture of the remains of food, filters rapidly as a thin fluid, either clear as water, or occasionally slightly opalescent, or somewhat greenish (in the latter case after standing a few days the green color is frequently increased on admixture with air). If the fluid which has become turbid from admixture with leukocytes and remains of epithelia, as well as from shreds of mucus—occasionally resembling the water with which

the mouth has been cleansed—is allowed to stand, a sediment forms which in more than fifty examinations of this kind I have rarely found to be higher than 4 to 5 per cent. of the total fluid. This sediment consists almost exclusively of mucus flocculi and nuclei of leukocytes whose protoplasmic ring has been digested, but also contains œsophageal and gastric epithelia which produce the turbidity. Now and then a few starch granules are noted, their amount being slight, so that in a test-tube, and on the addition of a few drops of Lugol's solution, the sediment in pure cases does not show a blue color. The specific gravity of the filtrate is, as a rule, abnormally low (about 1.004 to 1.008). The addition of iodin solution to the filtrate produces no change of color. Trommer's test is also negative, but the biuret test is, as a rule, positive. Upon polarization a rotation to the left of 0.2–1.0 per cent. is shown. That true gastric juice must contain hydrochloric acid is self-evident; the values for total acidity and for free hydrochloric acid, however, vary greatly. The amount of combined hydrochloric acid is, as a rule, not very high, and the amount of acid phosphates is usually less than in a trial breakfast. These are the conditions, provided there is simultaneously no obvious motor insufficiency. When this is not present in the "secretion from an empty stomach," we find no sarcinæ nor sprouting yeast, and in the incubation oven the fermentation test is also negative. In pure cases of the disease we find these conditions, immaterial whether the stomach has been thoroughly washed out the evening before or whether we have limited ourselves to prohibiting the patient from taking food after his supper. As Riegel emphasized, and as I demonstrated in quite a number of cases of hypersecretion, upon introducing the stomach-tube and removing the contents of the stomach at intervals of an hour or every two hours, we invariably observe a decided amount of secretion in the stomach although the patient in question has, in the meantime, entirely abstained from alimentary stimulation.

SYMPTOMS

From this peculiarity hypersecretion is accorded a special clinical position, as is its due also in a clinico-symptomatologic aspect, because the symptoms which bring the patient to the physician often present more or less uniformity. The patients upon prolonged duration of the disease and in spite of a good appetite are usually emaciated with tissues deficient in fluid (dry skin!), they complain of gnawing and painful sensations in the gastric region which are generally persistent, and—what is symptomatologically worthy of remark—these are present both night and morning, and are relieved by vomiting. The vomitus itself is usually a thin fluid, of acid taste, and often without admixture of food. In consequence of the loss of large amounts of gastric juice, there is, as a rule, thirst and constipation, and the urine on account of an increase of its alkalinity fre-

quently shows a phosphate sediment (gastrogenous phosphaturia). In typical cases the test breakfast reveals certain peculiarities which are conspicuous to the expert. There is a large amount (400 c.c. and more) of very thin, fluid, stomach contents which show relatively slight sediment (the layer quotient rarely amounts to more than 20 or 30 per cent.). The sediment has an extraordinarily fine, purée-like appearance, in consequence of the excellent amylorrhexis due to prolonged contact of the gastric contents and gastric juice. The trial breakfast generally filters very rapidly, and the watery filtrate upon addition of an iodin solution (in consequence of disturbed amylolysis) usually assumes a violet or more or less bluish color. The values for total acidity and for free hydrochloric acid vary. Not rarely —but by no means always—hyperacidity is present. In several cases of hypersecretion with simultaneous disturbance in motility I have noticed the peculiar circumstance that a few hours after the ingestion of one-half to one liter of milk into a stomach which was previously washed clean, an opalescent, turbid fluid without casein flocculi (which are otherwise present but here are digested with enormous rapidity) was obtained and at the upper border of the fluid sometimes a thin, at other times a thicker, coating of cream accumulated so that a well developed intermediary layer consisting of fluid and fat was formed.

DIAGNOSIS

In the diagnosis of hypersecretion the introduction of the stomach-tube upon an empty stomach is absolutely necessary, and in the diagnostic judgment of the contents which are evacuated from the empty stomach the constituents of the secretion which have here been described are important for the reason that the "residue" of the empty stomach may easily be confounded with the "secretion" of the same. The finding in the empty stomach of an appreciable "residue" which constantly shows a higher layer quotient than that of the "secretion" from the empty stomach, is always a sign of motor insufficiency. In those cases in which we must decide the question whether pure hypersecretion or hypersecretion combined with disturbance of motility is present, I advise as the first step in the differential diagnosis the administration of a teaspoonful to a tablespoonful of currants, the stomach having been washed out the evening before, also the microscopic examination of the gastric contents for micro-parasites, finally, the fermentation test conducted in an incubation oven.[1] A minute

[1] In the microscopic examination of the contents of the stomach in cases of hypersecretion with disturbance of motility, at a time when it should have been empty, I have occasionally found a remarkably slight amount of yeast present, and subsequently, a few times, chains of a delicate, thin, elongated variety of bacteria, as well as once distinct fungi mycelia which I also found in two cases of subacidity, and to which I attach no particular diagnostic significance.

investigation of gastric motility by the objective method of the currant test, and microscopic examination and fermentation in the incubation oven, appear to me to be particularly important because in the investigation of cases of hypersecretion the proofs whether a disturbance in motility is present or absent must be established. This differentiation is of vital importance for the clinical, especially for the theoretic, conception of cases, much more so than the otherwise justifiable classification of cases of hypersecretion into acute or chronic, into intermittent or relapsing, forms.

PATHOGENESIS

Disputes have arisen as to the pathogenesis of hypersecretion. The controversy above all has revolved about the question whether every case of hypersecretion is the result of pyloric stenosis expressed functionally or whether it is motor insufficiency. In truth, a number of authors look upon hypersecretion merely as a *retention* of secretion produced by motor insufficiency, basing their views upon the fact that the finding of motor insufficiency in cases of hypersecretion is not unusual, but even very frequent. Nevertheless, various authors (Riegel, Wilkens, Martius, Lichtheim, Gintl and others) have observed undoubted cases of chronic hypersecretion in which no objective symptoms of motor insufficiency could be detected, and I have reported two cases of this kind which led me to look upon hypersecretion as the expression of a chronic irritative condition of the secreting parenchyma in the etiology of which various factors play a part. The hypothesis that every hypersecretion is a simple retention of secretion, therefore, appears to me to be insufficient, because other authors as well as I have observed cases of motor insufficiency in which, after the disturbance in motility had been removed, the symptoms of hypersecretion persisted for days and weeks before they disappeared,[1] and because in a series of researches which I have not as yet published I demonstrated (in contrast to Boas) that under rectal nutrition—therefore when local stimulus from the stomach contents was entirely absent—the amount of secretion obtained from the empty stomach was just as profuse as during the time when food was introduced by mouth. For this reason I consider the nature of continuous hypersecretion to be a persistent irritability of the secreting parenchyma which itself may be due to a number of causes, among others the irritation of stagnant masses of food. However, as Riegel has emphasized there is in the latter a disproportion between the amount of irritation and the amount of secretion, therefore a special irritability of

[1] That such an irritation of the parenchyma may exist for some weeks after a successful gastroenterostomy I recently saw proven by the cure of a case of motor insufficiency due to perigastritis, which should be of deep interest because the perigastritis was the consequence of a laparotomy performed to recover a stomach-tube which had been swallowed by the patient (not while in the hospital).

the secreting parenchyma must be presupposed if the symptoms of hypersecretion become prominent. In regard to the etiologic importance of spasm of the pylorus which is occasionally observed in ulcer of the stomach —not rare in chronic hypersecretion—I limit myself to the remark that such spasms of the pylorus, under some conditions, may certainly lead to a retention of secretion in the stomach free from food, namely, if they have begun during a time in which the stomach contained no food. With the exception of ulcer of the stomach, which is frequent in hypersecretion, the pathologic anatomy of cases of hypersecretion has as yet taught us little regarding the pathogenesis of this morbid condition; for, in so far as exact investigation of the various areas of the stomach is concerned, the number of cases studied is quite scant. In a case reported by Myer and myself a few years ago there was but slight tortuosity and dissemination of the glands, also but slight change in the interstitial tissue. These findings were absent in a second case which I clinically observed. The literature, too, of the anatomical findings in cases of hypersecretion, which I cannot here describe, is contradictory. Nevertheless, in discussing the anatomy, I cannot refrain from stating that in cases of hypersecretion I have repeatedly observed gastroptosis, which, however, was usually the mechanical consequence of habitual overloading of the stomach with secretion, and also that, in those cases of hypersecretion in which an ulcer of the stomach existed, a permanent secretory irritation was perhaps due to the fact that exposed nerves upon the floor of the ulcer were irritated by the gastric contents or the gastric juice, and this, perhaps, produced inflammatory changes. Of the neurogenous type of hypersecretion those cases observed during gastric crises are the best examples. To-day, when we have learned from Pawlow's classical investigations to appreciate the relation of the nervous system to the secretion of the gastric juice, it hardly appears remarkable that hypersecretion may be of neurogenic origin. It is interesting, too, to note that hypersecretion is chiefly a disease of middle life, that it occurs more often in men than in women, and that it is more frequent in countries in which ulcer and hyperacidity are common than in other regions.

TREATMENT

The treatment of hypersecretion, primarily, is identical with the treatment of the underlying cause. Where there is a disturbance in motility, this must be combated with all the measures at our command. In those cases in which no disturbance of motility exists—which are decidedly the rarer—all the points come into question which have already been described under the treatment of hyperacidity, and here the plentiful administration of fat appears to be in order, since other authors, as well as I, have seen improvement and even cure follow its use in cases of hypersecretion. Concentrated solutions of sugar must be avoided, because the stomach in hyper-

secretion, in consequence of habitual overloading with secretion, already has a tendency to myoparesis. The latter factor, in combination with our purpose of frequently introducing acid-combining material into the stomach, shows the wisdom of administering small and frequent meals. For several reasons, those substances which increase the secretion of the gastric juice should not be employed as food. Alkalies are advisable when a hyperacid secretion causes spasm of the pylorus, as in the case of hyperacidity. Frequently, however, the pains are mitigated by the administration of other acid-combining substances, for example, eggs or milk. In the general diet, the amount of albuminous food, on account of the acid-combining properties of the albumin, should not be too small. If the palliative measures just mentioned are not sufficient, amelioration may often be produced by the subcutaneous administration of atropin, and Reichmann advises also the internal administration of not too minute doses of silver nitrate. In well developed cases of long duration we cannot get along without the introduction of the stomach-tube, which serves a particularly useful purpose in evacuating the surplus amount of secretion present at an improper time. Lavage of the stomach with water is only necessary when there is simultaneous disturbance of motility. When this becomes expedient, Riegel advises the use of an alkaline water, and Rosenheim, as well as Rost, praises irrigations with a 1–1000 solution of silver nitrate. Only when the measures that have been described prove inadequate, and absolute rest of the stomach for some time by rectal alimentation is ineffectual, does gastroenterostomy come into question. In those cases in which there is no disturbance in motility this operation has a curative effect by promoting the discharge from the stomach of irritating and burdensome secretions which prevent the healing of wounds that may be present, and by preventing the vomiting of food and gastric juice protects the patient from undernutrition and losses of chlorin. In some cases the treatment must also take into consideration the consequences of hypersecretion, for example, the gastroptosis, as well as the general loss or general dryness of tissue, but this is not the place in which to outline the treatment of these special pathologic conditions.

In reviewing what is here stated of the diagnosis and treatment of secretory disturbances of the stomach, the conclusion will be reached that the last quarter of the preceding century, in particular, has very materially added to our knowledge of these affections and broadened our sphere of action. Our present methods have been built upon a firm foundation, which may be more readily reviewed than was formerly the case. This development of an important diagnostic and therapeutic branch of gastric pathology is due not only to a generally recognized impetus given to medicine in the second half of the preceding century, to the methods of research and the exact results of scientific investigations, but, above all, to the

acumen and well-directed, energetic labors of those men who have made the stomach-tube the common property of the clinic and the practising physician (Kussmaul, v. Leube and others). The introduction of the stomach-tube, and particularly the introduction of the soft stomach-tube into medical practice—due to Ewald—was an event of almost historic importance, as is pointed out in another article in this volume. To acknowledge this, and to express it, can nowhere be more fitting than in concluding a description of a branch of medicine in which accurate knowledge and full comprehension have been made possible only by the use of the stomach-tube.

SUPPLEMENT

In this article on the analysis of clinical symptoms of individual secretory disturbances, it has been repeatedly intimated that an actual relation between definite, clinical symptom-complexes and definite disturbances of secretion cannot always be demonstrated. Therefore, it is still a mooted question in which cases the practitioner is justified in making an exact investigation of the gastric juice secretion by evacuating the gastric contents. This question can only be answered in a general way by the statement that a test of the gastric contents appears to be indicated in all those cases in which, without such examination, the diagnosis remains uncertain, and in which there are contraindications (in regard to this point see the article by Fleiner, pages 48 and 49) to the introduction of the gastric tube. As nothing of importance in the macroscopic investigation of the contents removed from the stomach can here be added to what was mentioned in the preceding articles, the method of *chemical* examination shall be briefly described: In each individual case we begin with dipping a piece of Congo paper in the filtered (and also in the non-filtered) gastric contents. The result of this examination indicates what further steps are to be taken in the investigation, since, according to what was said in the preceding article, if free hydrochloric acid is absent we ascertain by testing with litmus paper whether the gastric contents have an acid reaction, and, if this be the case, we examine for lactic acid. For the latter purpose I advise my modification of Uffelmann's process, since only pathologic amounts of lactic acid can be demonstrated after a test breakfast. This is carried out by using a graduated test-tube specially constructed for this purpose, which may be shaken, so that 5 c.c. of filtered gastric contents are shaken up with 20 c.c. of ether; the gastric juice and 15 c.c. of ethereal extract are allowed to flow off, and the remaining 5 c.c. of ethereal extract with 20 c.c. of water are shaken up with 2 drops of a 1 per cent. iron chlorid solution (liquor ferri sesquichlorati, 1–10). If lactic acid is present in a proportion of 5 to 1000 or more, the water turns decidedly green. If any doubt exists in regard to the lactic acid test, it is always advisable to investigate the gastric contents microscopically for an increase

of bacteria, especially the long bacteria commonly found therein. In those cases in which we are unable to obtain much material with the stomach-tube, the latter process should never be omitted, because the microscopic finding of numerous long bacteria in the gastric contents, as well as the presence or absence of the protoplasmic ring of leukocytes in those cases in which a chemical examination of the gastric contents cannot be made for want of sufficient material, may give us valuable information concerning the gastric juice secretions.

The total acidity is ascertained by the method Prof. Leo described in his article (which see), and phenolphthalein is generally used. When we suspect apepsia this may be advantageously supplemented by a comparative titration with tincture of cochineal (or with tincture of litmus). In the few cases in which we desire to determine the combined HCl, Leo's method is advisable. If, for any reason, we wish to ascertain the quantitative amount of free hydrochloric acid, a few drops of a one-half per cent. alcoholic solution of dimethylamidoazobenzol is added to a measured quantity (10 c.c.) of filtered gastric contents and titrated with a decinormal solution of soda until the red color disappears; or we may use the dipping method with Congo paper. When employing the latter the platinum loop is advised for withdrawing small amounts (Ewald), and the best way of proceeding is to compare the drop taken from the gastric contents with a drop of water of the same size placed upon Congo paper. In case Linossier's reagent is not used from the onset (dimethylamidoazobenzol 0.25, phenolphthalein 2.0, alcohol 100.0), after the amount of free hydrochloric acid has been obtained a few drops of an alcoholic solution of phenolphthalein is added, and the total acidity is determined by the same test.

Mett's process for the quantitative estimation of pepsin, which may under some circumstances be of practical use in the diagnosis of apepsia gastrica or for the topical diagnosis of gastric carcinoma, is best carried out in daily practice in the following way: 10 c.c. of the filtered anacid or subacid gastric contents, to which one c.c. of a 2 per cent. solution of hydrochloric acid which is kept on hand should be added, are placed in a small glass bottle with a beveled cover, or in some other low and proportionately wide glass vessel closed with a glass stopper. With an albumin tube, prepared according to the method described upon page 92, well washed for 24 hours before use, and with the aid of a magnifying glass, we determine the total amount of albumin digested during this time. According to my experience (and when the test is made in the manner described), in the normal person this rarely amounts to less than 5 mm., sometimes, however, to decidedly more (occasionally to double this amount). Schorlemmer (*Archiv für Verdauungskrankheiten*, Bd. VIII, Heft 3 und 4) has recently advised an ingenious modification of Mett's method of examination, but it would be well first to decide by comparative investigations whether the additions to improve the process are absolutely necessary in daily prac-

tice. The dilution recently proposed by Nirenstein and Schiff (*Archiv für Verdauungskrankheiten,* Bd. VIII, Heft 6) with a 1-20th normal hydrochloric acid solution in the proportion of 1 to 16 for those cases in which we have pronounced the estimations of pepsin as valuable—cases of *extreme* subacidity—is unnecessary, as in these cases, particularly, the results of the diluted and non-diluted test appear to be almost exactly alike.

The estimation of lab-zymogen in accordance with the method proposed by Boas is best made in the following manner: 2 c.c. of gastric juice (with *high-graded* secretory insufficiency a neutralization of the acid by alkalies is only necessary when lactic acid is present and—to prevent a greater dilution—this is best done with a normal, not a decinormal, soda solution) are placed in a small measuring glass and diluted with 8 c.c. of water and 5 c.c. of milk (this must not be of acid reaction) mixed in a test-tube (dilution of 1 to 10). The remaining 5 c.c. are mixed with 5 e.c. of water so as to produce 10 c.c., and 5 c.c. of this mixture are added to 5 c.c. of milk (dilution of 1 to 20). The 5 e.e. remaining from this test are added as before to 5 c.c. of water, and one-half of this is poured off and mixed with 5 c.c. of milk (dilution of 1 to 40). By repeating this process we produce dilutions of 1 to 80, 1 to 160, and 1 to 320. After the addition to each of one c.c. of a one per cent. calcium chlorid solution, the entire series is placed in the incubation oven, heated to 37° C., and allowed to remain for half an hour. Under these conditions the filtrate from a stomach which functions normally will coagulate milk in a dilution of 1 to 80 or even 1 to 160. Glässner (*Berliner klin. Wochenschr.*, 1902, Nr. 29), reports that the normal amount of lab-ferment in the gastric juice obtained by a test breakfast is 1 to 100, i. e., 0.1 c.c. of neutralized gastric juice will, within half an hour, caseate 10 c.c. of milk at a temperature of 30° to 40° C.

For further details of the methods of examination, the reader is referred to recent text-books upon the diagnosis and therapy of gastric diseases, especially to those of Boas, Ewald, Fleiner, Riegel, and Rosenheim, as well as to the text-books upon microscopieo-chemical diagnosis by v. Jaksch, Lenhartz and others. A critical compilation of the literature up to the year 1892 concerning the various methods of examination of the gastric juice is found in the monograph by Martius-Lüttke: "Die Magensäure des Menschen," Stuttgart, F. Enke, 1892.

DIAGNOSIS AND TREATMENT OF GASTRIC DILATATION

By F. RIEGEL, Giessen

CLINICAL FORMS

When a patient presents himself to us and states that he suffers from frequent vomiting of large residua of food, and that he repeatedly finds remains of food that he had consumed several days before, when, upon examination of the patient, the lower boundary of the stomach is found below the umbilicus with an approximate normal position of the upper, and when, throughout this entire region, a loud succussion sound can be produced, the suspicion of dilatation of the stomach is obvious. If, in such a case, seven hours after a test-meal, profuse quantities, a liter or more, are evacuated from the organ, and if an evacuation in the morning upon a presumably empty stomach shows profuse remains, we speak of *gastric dilatation,* or *gastrectasis.*

Two factors are necessary for the diagnosis of gastric dilatation—a *decided increase in the size of the organ* and an *insufficient motor activity,* a *so-called motor insufficiency.*

I do not agree with those modern investigators who recently have repeatedly declared that the terms "ectasis" and "dilatation of the stomach" are no longer expressive of present scientific views, if they intend to eliminate the term "gastric dilatation" from pathology, and to put in its place "mechanical insufficiency, myasthenia gastrica, ichochymia, motor gastric debility" or the like, which denote exclusively insufficient motor activity. These authors are certainly correct when they declare that the size of the stomach is no measure of its motor activity. A stomach may be larger than normal, yet, notwithstanding, have sufficient motor power, and another stomach may be well within the usual physiologic limits, and, nevertheless, show motor insufficiency. Boas is, therefore, certainly right when he says that we drift from a positive foundation in diagnosis when we make the limits and size of the stomach a criterion in deciding upon an existing gastric dilatation. But no conscientious physician will to-day diagnosticate gastric dilatation upon the basis alone of a large extension of the gastric limits.

The stomach may have suffered in its elasticity, may be more distensible, yet, nevertheless, still be capable of propelling its contents at the proper

time. If such a stomach be inflated with air or carbonic acid, it reaches far beyond the normal limits; such a stomach is abnormally elastic; nevertheless, it may be able to propel the ingesta at the right time like a normal stomach. This condition is designated *megalogastria.*

Such cases are discovered purely by accident; for megalogastria, as such, does not give rise to symptoms. The conception of ectasis demands more than an increase of size, more than abnormal elasticity; above all, it is necessary that the stomach should be unable to propel its contents at the proper time, that during the long period in which a normal stomach receives no food, more or less profuse contents are still within its walls.

We are forced to admit that the designation "gastric dilatation" primarily implies only a larger extent of space. Nevertheless, this same term "dilatation," in a certain sense, also indicates a condition of motor insufficiency, for, from what other cause has the patient with gastrectasis a dilated stomach, wherein is the root of his difficulties except in this, that the stomach is incapable of propelling its contents, and is, therefore, constantly overfilled! Even the older physicians in their conception of gastric dilatation not only held the view of an abnormally large extension, but also that the stomach lacked the power of timely propulsion of its contents. That stomach alone is *permanently* dilated which cannot empty itself at normal periods.

If this view of gastric dilatation be accepted—and formerly it was universal—there is absolutely no reason for discarding the old, established designation "gastric dilatation." It is true that the determination of the size of the stomach alone is not sufficient, it is also necessary to ascertain the motor activity of the dilated organ. At the bedside it is never satisfactory to determine the physical changes only of the diseased organ; we must also picture to ourselves the disturbed function.

Similarly, we speak of a dilatation of the heart; but as, in the heart, the determination merely of an increase in size is not sufficient, so, in every case, we study the effect of this dilatation upon the circulation, both in the arterial and venous system, since increase in size is by no means always of like importance; this is also true of the stomach. In the conception of dilatation, a decrease in the motor power is included as well as increase of size. At the onset, however, it is clear that it makes a decided difference whether a stomach shows only decreased motor power, or whether it simultaneously shows a decided and permanent increase in size.

It must be admitted that formerly the error was frequently made of diagnosticating gastric dilatation solely because of an increase in size. But to-day no conscientious physician, upon suspicion of gastric dilatation, will limit himself only to determining the boundaries of the stomach. This would be just as one-sided and wrong as to make a diagnosis of gastric disease from the determination of the acidity alone, or to diagnosticate carcinoma from the absence of free hydrochloric acid alone. *The increase*

in size primarily awakens the suspicion of dilatation, but this is insufficient warrant for a diagnosis.

When there is a decided distention of the stomach combined with subjective symptoms, the physician should not neglect to introduce the stomach-tube, to determine the functions of the stomach in regard to motion and secretion.

The name "*motor insufficiency*" indicates only a *disturbance in function* which may occur in the most varied diseases, and is manifest when the gastric contents are not expelled at the proper time. This may occur in a dilated stomach, in a normal one, and even in a stomach decreased in size. The designation "motor insufficiency" in place of "dilatation" cannot be regarded as correct, for the reason that motor insufficiency is possible without any enlargement of the stomach. It is, however, a matter of consequence, whether, in the given case, the motor insufficiency has led to a permanent abnormal distention of the stomach or not, but only in the latter case do we speak of *dilatation*.

Atony is a special form of motor insufficiency. By this we mean a condition in which the tonus of the gastric musculature is diminished, in consequence of which there is delay in propelling its contents, without the boundaries of the stomach having exceeded their normal limits. Atony *may* lead to dilatation, but need not necessarily. When the latter is the case, we speak of *atonic dilatation*. The atonic stomach propels the gastric contents more slowly than is normal, but does finally propel them.

We must, therefore, differentiate between motor *insufficiency*, *atony* (a special variety of motor insufficiency), and *dilatation*. Megalogastria does not here come into question.

Before proceeding to the clinical picture of dilatation—and I purposely speak of the clinical picture of dilatation, and not of the disease "ectasis," for it is not a disease, *sui generis*—I thought it necessary to explain the conception of the term. On this point, as a glance at the literature of recent years will show, there is a great difference of opinion.[1]

An attempt has been made to substitute a different designation for the term in general use, "dilatation." If the conception "gastric dilatation" is considered in the above mentioned sense, there is no reason for the introduction of new terms, as they have no advantage over the old. Naturally, in determining a dilatation we must not limit ourselves to the mere estimation of the size, but we must endeavor to investigate the intensity of the disturbance in function in connection therewith.

There are varying *degrees* in the nature of the clinical picture which we designate by the term "ectasis," but no well-defined limit from which we may speak of dilatation and from which we should speak of motor

[1] I refer particularly to Hesse, "The Conception of, and the Term, 'Gastric Dilatation,' in German Literature since 1875." *Berliner klin. Wochenschr.* 1900, Nos. 23 and 24.

insufficiency and atony. The diagnosis of gastric dilatation is easy when the stomach permanently contains decided residue of food, and when its boundaries, therefore, far exceed the normal limits. In milder cases the condition is different.

No stomach, not even a normal one, *constantly* shows the same distention; its boundaries vary, we may almost say hourly. The same is true of the dilated stomach, and also of the stomach with motor insufficiency, which, although it finally expels the ingesta, does this decidedly more slowly than the normal organ. The stomach with motor insufficiency is *relatively* dilated in so far that it still retains ingesta at a time when a normal stomach has already expelled it.

In this sense we speak of *relative dilatation* if a stomach, seven hours after a test-meal, still contains considerable remains of food, while its limits do not exceed the normal boundaries. For, at this time, the stomach should be empty and markedly contracted. In such cases we do not make a diagnosis of gastric dilatation, but only when the lower and lateral borders of the stomach permanently exceed the normal boundaries, and when the stomach *permanently* retains ingesta. In spite of this, in the first mentioned case, there is a relative and *temporary* increase in size, and there can be no doubt that, upon prolonged duration and long-continued action of these deleterious causes in such a dilated stomach, true dilatation may gradually develop.

It is, therefore, evident that there is no sharp dividing line between dilatation and motor insufficiency. Dilatation naturally includes motor insufficiency, for the stomach can only be dilated when its contents are not entirely expelled at the right time. Every true dilatation, therefore, must be accompanied by motor insufficiency; but not every insufficiency need lead to true ectasis. It is obvious, therefore, that in discussing dilatation we also consider motor insufficiency and atony.

As to the *causes* of dilatation, it must be remembered that this can only develop upon a basis of motor insufficiency. If the stomach cannot discharge its contents fully and at the proper time, it must be distended longer than is normal, it must remain abnormally weighted. Gradually, decided distention occurs, and, finally, true ectasis.

Motor insufficiency can only arise *where there is a disproportion between requisite labor and the capacity for labor, where requisite labor and power of labor no longer, as in the norm, maintain an equilibrium.* When the motor power of the stomach is no longer commensurate with the demands made upon it, the work is only imperfectly done or after a long period; i. e., the stomach will expel the ingesta only partially or after a prolonged period of time. The causes of this diminished motor activity are various, and may be expressed as follows: 1. The amount to be propelled is abnormally large, as, for example, in acute, or prolonged, or frequently repeated over-distention of the stomach; 2. The expelling power, the muscular

power of the stomach, is deficient; 3. There is an abnormal resistance to the expulsion of the ingesta from the stomach.

While, in the group first mentioned, we are dealing with a direct damage or diminution of the motor power, in the third group, that of mechanical hindrance to the expulsion of the ingesta, we often find the muscular power increased, the muscularis even hypertrophic the same as in stenosis of a valve of the heart, that portion of the heart anterior to the stenosis is not only dilated but, at the same time, it becomes hypertrophic. Nevertheless, it is incapable of increased labor. This group represents by far the greatest number of dilatations, and includes the *stenoses of the pylorus and its surroundings.*

Much rarer is insufficiency, the *atony and atonic dilatation* which go hand in hand with flaccidity of the muscularis. Moderate degrees of atony are not rare. Such an atony may arise when the stomach either temporarily or for a long time has an unusual amount of work to perform. Thus, we see atony as an accompanying symptom of various affections of the stomach. We observe it particularly after acute distention of the stomach with food difficult to digest, we see it in persons who eat hurriedly and abundantly, and chew their food insufficiently. True dilatation may arise in consequence of such over-distention; *in children,* particularly, *acute dilatation* not rarely occurs in this manner. Occasionally it increases to an extreme, and not seldom such acute dilatations have a lethal outcome. In the latter case, other factors besides an acute distention of the stomach may play a rôle. Thus Kussmaul [1] many years ago called attention to the fact that in gastric dilatation all the symptoms of complete closure may occasionally appear and continue until large amounts of the stomach contents are expelled by vomiting or by lavage.

As Kussmaul pointed out, such cases cannot be due to a paralysis of the muscular wall by immoderate distention, for peristalsis of the stomach is seen to be markedly active when the stomach contents can no longer be propelled. Kussmaul believes that in cases of this kind a narrowing from tug and tension occurs at the point where the horizontal portion of the duodenum leads into the vertically fixed part at the vertebral column.

This conception of the combined action of a mechanical hindrance has gained decided support by the recent investigations of Albrecht [2] regarding "Arterio-Mesenteric Intestinal Occlusion at the Duodenal Jejunal Boundary and its Causal Relation to Gastrectasis." Albrecht has proven that the cases of acute gastric dilatation, above all with symptoms of ileus (intestinal obstruction), often have their origin in an arterio-mesenteric invagination of the inferior transverse portion of the duodenum, in that the stomach, as it dilates more and more, forces the transverse colon and the

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[1] "Peristaltic Unrest of the Stomach." *Volkmann's Samml. klin. Vortr.* Nr. 181.

[2] *Virchow's Archiv*, CLVI.

loops of the small intestine downward toward the pelvis, the mesentery, and particularly the arched fold under which the duodenum enters into the jejunum, becomes tense, and thus a constriction is caused in the mesenteric artery at the duodeno-jejunal boundary. Albrecht quite properly states that the vicious circle which is formed by the downward sinking of the folds of the small intestine causes tension in the mesentery which completes the compression of the duodenum. Thus the contents of the duodenum become more and more engorged, and the stomach constantly more full and dilated. The horizontal portion of the duodenum becomes still more overloaded, and the downward displacement of the colon and the small intestine still further increased.

That an acute atony of the stomach, an acute gastric dilatation, may favor the development to a high degree of an arterio-mesenteric duodenal incarceration is clear. In other cases, the compression of the duodenum by the mesentery of the small intestine may have been the beginning of the disastrous chain. In incomplete closure, and with an incomplete dislocation of the duodenum, there may be chronic dilatation with vomiting of bilious masses.

In cases of this kind recently reported, the symptom-complex was observed after chloroform narcosis preceding various operations, among them some upon the biliary passages. Bäumler,[1] in reports of two cases belonging to this category, has recently stated that numerous factors favor this form of intestinal immobility, and he groups these as follows: 1. Greater length of the mesentery of the small intestine; 2. Gastroptosis; 3. Enteroptosis in its influence upon the position of the duodenum and upon the root of the mesentery; 4. The position of the duodeno-jejunal boundary in relation to the vertebral column; 5. Marked lumbar lordosis; 6. High-graded emaciation and debility from preceding disease; 7. Chronic gastric dilatation in consequence of congenital or acquired stenosis of the pylorus; 8. Prolonged dorsal decubitus after operations; 9. Very complete emptying of the intestine prior to an operation.

Further predisposing factors for the development of atony in general are preceding severe infectious diseases, general constitutional affections, anemia, chlorosis, and the like.

Those who are poorly nourished, or debilitated by sexual excess or other causes, are highly susceptible to atony. Abnormal position of the stomach, gastroptosis, enteroptosis, extreme flaccidity of the abdominal walls may be designated as predisposing factors, even although they do not in themselves cause atony.

In some cases, the so-called weak, atonic stomach is *hereditary*. Frequently, the nutrition habitual from childhood may here play a rôle. Like every muscular organ, the stomach, from childhood, should be accustomed

[1] *Münchener med. Wochenschr.* 1901, Nr. 17.

to proper food, as this develops its muscular activity. Monotonous diet which is deficient in residue may so weaken the stomach that when, under unusual circumstances, demands are made upon it which can hardly be designated as harmful, its muscular tonus suffers and reacts by becoming flaccid.

At the onset, we might suppose that disturbances in the secretion of the gastric juice would decidedly influence the propulsion of the gastric contents, and thus indirectly cause disturbances of motor activity. Clinical observation shows that this is not the case to the extent, and in the manner, that we might be inclined to assume. Such a reaction we should most look for in pure *achylia* in which the secretion of gastric juice is entirely absent. But here, particularly, such a disturbance does not appear; on the contrary, the motor power is good throughout, and even increased. This is best explained by studying the remarkable results of the experiments of Pawlow [1] in which he proved that the duodenal mucous membrane regulates the propulsion of the gastric contents into the intestine, regardless of the condition of fulness in the latter, from the fact that it depends upon the reaction of the gastric contents and their acidity. Alkaline contents usually pass from the stomach very soon. On the other hand, after the intestine has received a portion of the acid gastric contents, it causes by reflex action a temporary closure of the pylorus. The acid food mass, which the pylorus has permitted to pass through, causes an increased secretion of pancreatic juice, and by and by becomes neutralized. Only after this has occurred is the expulsion of further acid masses from the stomach permitted, and, accordingly, the quickened motor activity of the stomach in achylia is readily understood.

On the other hand, in cases of increased gastric juice secretion, particularly in *hypersecretion*, we not infrequently see disturbances of the motor activity in the form of delayed expulsion of the ingesta, and not rarely also of ectasis. The connection between these is a varying one. Many authors regard hypersecretion in all cases as secondary, as the consequence, not the cause, of ectasis. That hypersecretion and ectasis are frequently observed in combination is certain; which is primary, and which secondary, cannot be so readily decided. Even where an organic pyloric stenosis can be determined, this does not prove that the mechanical obstruction is the cause of the hypersecretion. Pyloric stenosis in which there is a sufficient secretion of gastric juice by no means always leads to hypersecretion. That hypersecretion often rapidly disappears after gastroenterostomy, which so greatly facilitates the flow of the secretion, cannot be regarded as a proof that the latter is a consequence of the stasis. Prolonged stasis may keep up the irritative condition, its disappearance may

[1] "Das experiment als zeitgemässe und einheitliche Methode medicinischer Forschung." Wiesbaden, 1900.

remove it, and facilitate the outpouring of the secretion; but stenosis in itself does not explain the prolonged secretion of gastric juice.

The secretion of gastric juice normally is not continuous, but periodic. As Pawlow [1] first showed by conclusive experiments, the normal stomach, empty of food, does not secrete gastric juice. It only secretes it upon *digestive*, not upon mechanical, stimulation. For this reason, therefore, it is not to be expected that stenosis in itself will cause a continuous secretion of gastric juice. If the stomach of a patient suffering from hypersecretion is washed perfectly clean, and is evacuated after a few hours, during which time the patient has taken no food, a greater or less amount of secretion is nevertheless found. The stomach therefore, despite the absence of any digestive stimulation, has spontaneously secreted gastric juice. This certainly favors abnormal stimulation. On the other hand, we also see, although not so frequently, cases of hypersecretion without pyloric stenosis and without actual dilatation. It is true these cases are readily overlooked. Hence it follows that pyloric stenosis in itself is not the primal, and not the only, cause of hypersecretion. Inversely, however, it is doubtless true that hypersecretion alone may lead to dilatation by limiting the transformation of the amylacea to such a degree that upon prolonged retention fermentation of gas is produced, and, finally, if this continue for some time causes dilatation. According to Pawlow's investigations, the abnormally acid contents and the profuse amount of acid-secretion remain in the stomach a much longer time than the gastric contents which are normally acid. The transformation of the acid gastric digestion to the alkaline intestinal digestion by the pancreatic juice must be slow and difficult in those cases in which a much higher degree of acidity and a much larger amount of acid gastric juice is present than in the normal, as is the case in hypersecretion.

Particularly in the case of hypersecretion, spastic proeesses at the pylorus are observed which inhibit the exit of the acid ingesta; this, accordingly, can only be regarded as a proper preventive, as a protection against a disturbance of digestion of the small intestine. These factors are calculated to produce a long stagnation of the ingesta even without the existence of a stenosis. Hypersecretion in itself must cause stasis, and may, therefore, in itself finally produce dilatation. Hence it cannot appear strange that, particularly in the case of hypersecretion, a change in the symptoms is observed, sometimes improvement, sometimes aggravation, sometimes an increase of the motor insufficiency.

It has been maintained that marked *psychical* influences give rise to such atonies. That psychical influences may affect the secretion of the gastric juice and also the motility of the stomach is no doubt true. But these are only transitory disturbances. It has not yet been proven that chronic ectasis may develop upon a purely nervous basis.

[1] "Die Arbeit der Verdauungsdrüsen," 1898.

Whether, and in how far, diseases of the *central nervous system* lead to atony and dilatation has not yet been determined with certainty. Isolated, recent observations appear to favor the possibility of the development of gastric dilatation from central influences.

The cases are not rare in which patients with gastrectasis refer the beginning of their disturbances to a *single trauma* in the gastric region. As is shown by careful analysis of these cases, many of the ectases are caused by a stenosis of the pylorus or in its vicinity. This stenosis in a number of cases originates in a cicatricial narrowing brought about by necrosis and ulceration due to trauma; in other cases the trauma first causes a perigastritis, and, in consequence of this, adhesions to the neighboring organs; when these adhesions in their further course affect the region of the pylorus, they also lead to stenosis. These traumatic stenoses have the peculiarity that the stenotic symptoms never develop at once, but often only a long time after the trauma.

Simple *atony* may also develop after trauma; it has been noted after operations upon the abdomen. In contrast with the form just mentioned, atonies after traumata mostly develop suddenly and soon after the injury; they may even themselves prove fatal, as in a case recently reported by Grundzach.[1]

Not only a single trauma, but also *frequently repeated ones* which affect the gastric region play a rôle in the etiology of ectasis.

Many occupations necessitate that the labor be performed in a position which causes the body to be bent forward, that the epigastrium be exposed to the pressure of tools which are stemmed against it, and thus subject it to long-continued pressure. This may cause circulatory disturbance, a pressure ulcer, but also cicatricial stenoses or perigastritis, and thus lead to gastrectasis. But we cannot assume a cicatricial stenosis in every case of gastrectasis; even active peristalsis of the stomach is no positive proof. At all events, in a number of dilatations developed in this manner, we note decided variation in the symptoms, and, under some circumstances, even complete retardation, so that the assumption that spasms of the pylorus are chiefly the cause does not appear unjustifiable.

It has been maintained that some *narcotics*, such as alcohol and tobacco, have a relaxing and paralyzing effect upon the muscularis of the stomach. No cases developing in this manner have as yet been determined with certainty.

Although it cannot be denied that gastrectasis may develop upon the basis of atony, nevertheless, at the onset, in every dilatation of extreme degree the suspicion is justified that a mechanical hindrance, or an organic stenosis either at the pylorus or in its vicinity, is the cause of the dilatation. *Mechanical hindrances are by far the most frequent causes of ectasis,*

[1] *Revue de Médecine*, Mars, 1899.

and the most important of these are *cicatrices from ulcers, carcinoma,* and *cicatrices from caustics.*

The consequences of dilatation will naturally also appear when the obstruction which causes stenosis is situated somewhat lower down, below the pylorus and in the beginning of the duodenum. *Foreign bodies* which find their way into the pylorus, cherry stones, medlar stones, invaginated gall-stones, have caused dilatation there or in the duodenum. Quite exceptionally, cases of tuberculous stenosis of the pylorus have been observed. Jacobi [1] has recently reported such a case. Very rare causes of pyloric stenosis are benign tumors, pediculated polypi, connective tissue hypertrophy of the pylorus, external tumors pressing upon the pylorus (large gall-stones in the gall-bladder, Minkowski).

Pyloric stenosis is rarely *congenital;* nevertheless, quite a series of reports are at hand in which a decided hypertrophy of the pylorus caused marked symptoms of stenosis in nurslings. It is true a number of cases of supposed congenital pyloric stenosis admit of a different explanation. Pfaundler,[2] particularly, has called attention to the fact that recently in examining cadavers of nurslings, in about every third case the stomach was found in a peculiar state of persistent, muscular rigidity. The pylorus itself, in consequence of the permanent contraction, was very narrow, and frequently did not permit the passage of a medium thick sound. Pfaundler believes that many cases reported as congenital pyloric stenosis correspond minutely with the findings in systolic stomachs of this kind. However, he does not deny that there are actual clinical pictures among nurslings which point to an existing pyloric stenosis.

These are the most important causes of dilatation and motor insufficiency. The *degree* is a varying one. High-graded ectasis is observed in organic stenosis, among which by far the most frequent causes are carcinoma and cicatrices from ulcers.

PATHOLOGY

Anatomically, the picture presented by ectasis is in proportion to the cause and duration of the dilatation, the degree and nature of the stenosis. Motor insufficiency, as such, so long as it does not give rise to dilatation, is not susceptible to anatomical diagnosis, or, at most, only in the rare cases with cirrhotic change or diffuse malignant degeneration of the stomach.

The degree of dilatation varies; primarily, and chiefly, the fundus and the greater curvature which bear the brunt of the affection are involved in the dilatation. But, in severe grades, the dilatation is more general, chiefly lateral, as may often be determined *intra vitam.*

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[1] *Wiener klinische Wochenschrift,* 1900, Nr. 48.

[2] *Ibid.,* 1898, Nr. 52.

In isolated cases of ectasis the walls of the stomach are very differently affected; sometimes they are thinned, in other cases they show decided thickening. This variation depends primarily upon the cause of the dilatation, and, secondly, upon the duration of the affection.

Decided hypertrophy, particularly of the pylorus, is seen chiefly in stenoses of the pylorus and of its surrounding areas. The muscularis is often three or four times thicker than the norm. In other cases there is no hypertrophy of this kind; on the contrary, the muscularis appears thin and atrophic as in the case of atonic dilatation; not infrequently atrophy, and fatty and other forms of degeneration of the muscularis are noted in the later stages of long-existing ectasis.

The relation of the sub-mucosa and mucosa depends much less upon the dilatation itself than upon the underlying causes of the ectases. But in regard to these points we must refer the reader to the individual forms of disease which produce dilatation.

SYMPTOMATOLOGY

In the description of the clinical picture, "ectasis," I shall limit myself entirely to the symtomatology of dilatation. It would be too great a digression if I were to consider all varieties which might secondarily cause dilatation, such as carcinoma, cicatrices from ulcers, perigastric adhesions, chronic gastritis, hypersecretion and the like. Here we will consider only the clinical picture of dilatation as we meet it in daily practice. It is true the diagnosis, "ectasis," is an imperfect one; it tells us only that the stomach is dilated, and that motor insufficiency is present; our object must be to determine the cause, and, as a rule, this offers no insurmountable difficulty.

The cases are comparatively rare in which the special diagnosis of the cause of the dilatation is difficult. Often the examination of the patient not only suffices for this but enables us to determine the underlying affection which produced it. Naturally, there are cases in which prolonged observation and the most minute investigation of the course of the disease and of the results of treatment are necessary; but at the bedside we should first determine the dilatation, and then seek for its cause.

The symptomatology naturally varies with the degree of the dilatation. Mild grades of ectasis produce less prominent symptoms than higher grades. As I have already stated, there is no sharp division between motor insufficiency and dilatation, in spite of many attempts to define it. It is impossible to draw a line, from which point the stomach may be regarded as dilated. This might be feasible in an organ always of absolutely the same size, but not in an organ which, like the stomach, shows a varying extension at different periods. Naturally there are extreme degrees of dilatation which enable us to say, without more ado, that the stomach is decidedly

dilated. Slighter degrees are designated by one author only as motor insufficiency, by another, as dilatation. In estimating the size of the stomach, we must consider the period as well as the absolute degree of distention.

In no group of diseases is the *time of the physical examination* so important as in gastric affections. It is by no means immaterial at what time of the day we examine a patient suffering from gastric disease. If, early in the morning before food is taken, we can elicit a distinct splashing sound, and so prove that large quantities of fluid and air are still in the stomach when it should be empty, we decide at once that motor insufficiency is present. In a certain sense we may also consider this stomach to be dilated although its lower boundary does not extend below the umbilicus. For it is at least temporarily dilated; i. e., more than normally distended; early in the morning it should be empty and contracted. We may, therefore, speak of relative dilatation in contrast to absolute and extreme dilatation. In practice, however, it is necessary not only to determine the extreme grades, but also the slighter forms of dilatation and motor insufficiency.

The earlier the stage in which the diagnosis is made the greater the chances of controlling the malady. But, as I should like to emphasize at this point, to obtain a clear insight we must employ all methods in the examination. A well-trained practitioner will sometimes, on first observing the abdomen, be able to diagnosticate gastric dilatation just as we diagnosticate tabes dorsalis from the gait, and aortic insufficiency from the pulse. But no conscientious physician would be content with this. For the name, alone, of the disease, does not suffice, but, at the bedside, we must fully comprehend the nature and intensity of the physiologic disturbance of function, and for this purpose we require numerous methods of investigation.

If this had always been borne in mind in making the diagnosis of ectasis, we should not have erred by regarding the size of the stomach as of most importance, and of considering this alone the decisive factor. But it is just as partial and extreme to determine the degree of ectasis from the amount of material evacuated or from the amount of residue, and to consider the size of the organ as entirely secondary. They are related to each other, and still further methods of investigation must be resorted to if certainty in diagnosis is to be assured, as well as a full understanding of the nature and intensity of the disturbances in a given case.

To begin with the **subjective symptoms**, it must be remarked that these vary not alone according to the degree of the dilatation, but also according to the cause and the manner of its development. As a rule the first symptom of which the patients complain is a feeling of tension, fulness, and pressure after meals. Some experience abnormal sensations of this kind even after light meals; some report that they feel better in the recum-

bent posture than when standing, particularly after a somewhat heavier meal.

Others complain of eructations and pyrosis, others again of a sensation of active unrest in the stomach which sometimes increases to actual spasmodic pain. Occasionally these disturbances are relieved by vomiting. This is observed chiefly in pyloric stenosis with sufficient or increased secretion of gastric juice; but spastic conditions of the pyloric ring may also of themselves produce painful spasmodic attacks.

The *appetite* is capricious; as a rule it is decreased; only rarely, in mild cases is it normal or increased. Often the patients have a feeling of satiety after partaking of small amounts of food; bulimia occurring paroxysmally is rarely complained of.

The *thirst* is more important than the state of the appetite. In almost all well marked cases there is complaint of thirst. The more extreme the degree of dilatation and motor insufficiency, the more markedly is thirst increased. The stomach itself does not absorb water, but, on the contrary, a more or less active excretion of water occurs in the stomach with the absorption of many substances, such as sugar, dextrin, alcohol, peptone, etc.; with well marked dilatation of the stomach the propulsion of fluid becomes exceedingly difficult. This remarkable increase in thirst is readily explained; and the fact that in dilatation more fluid is frequently found in the stomach than is consumed is comprehensible. The decreased absorption of water corresponds with the decreased excretion of urine, to which we shall recur in describing the objective symptoms.

The state of the *tongue* is not characteristic; in the more marked cases there is a noteworthy tendency to dryness. In general, the appearance of the tongue varies; but it is by no means, as was formerly supposed, a reflection of the stomach.

An important and, probably, the most important symptom of dilatation is *vomiting*. In the milder grades of motor insufficiency it may be entirely absent; in well marked ectasis it is almost always present, naturally, in varying frequency. In some cases its peculiarities at once suggest dilatation. Thus, if the quantity of the vomited material exceed the quantity of the last meal, if remains of food which had been taken previously, often some days before, be detected in the vomitus, motor insufficiency is indicated.

The patients are usually decidedly relieved by vomiting. For this reason they often attempt to produce it voluntarily. Vomiting may be repeated daily; frequently it occurs at very irregular intervals.

The appearance of the vomited material varies greatly; the differences depend chiefly upon the underlying affection which causes the dilatation and upon the secretion of gastric juice. The vomitus in dilatation with hypersecretion has a different appearance from that due to carcinoma of the pylorus. In the former case we observe the well known three layers:

the lower layer consisting of fine remains of starch, the next of a decidedly acid fluid, and the upper layer being frothy as the expression of gas fermentation; in the latter case coarse remains of food are seen, particularly remains of meat, with an absence of free hydrochloric acid, and a decided reaction for lactic acid. I cannot here describe the special peculiarities of the vomited material, which differ according to the underlying affection that causes the dilatation. I must refer the reader to the individual diseases themselves.

An admixture of blood in the vomitus is not rare; this is most frequently seen in carcinoma, but it also occurs in other processes. Naturally this finding has nothing to do with the dilatation as such. Moderate degrees of motor insufficiency often exist for a long time without impairing the general condition. Patients with high-graded ectasis, however, as a rule, are feeble, weak, and incapable of exertion. We commonly meet with complaints of numbness in the head, of headache and of vertigo.

The *bowels,* as a rule, are constipated, occasionally not. Very rarely do the patients complain of diarrhea; this is most frequently observed in stenoses with marked gaseous fermentation.

These most important symptoms of the patients, as will be noted, show but slight peculiarities. The **objective symptoms** are much more important, and are alone decisive in diagnosis. Naturally they also differ according to the degree of the dilatation. Slight grades of atony and motor insufficiency at first, that is, if the duration has not been too prolonged, do not materially affect the *general condition and nutrition.* It is different in high-graded, long-continued dilatation. Here the nutrition suffers decidedly. The patients are more or less emaciated; the fatty layer has disappeared, the skin is dry and withered, it may be raised in large folds and desquamates; the muscles are thin and atrophic. This picture is met with not only in cases of dilatation due to carcinoma, but is also observed in dilatation occurring in benign affections, and, naturally, it depends upon the duration of the disease. Carcinomatous dilatation leads more rapidly to loss of strength than the benign forms. But, in any given case, the appearance, the degree of emaciation, cannot be taken as positive indications of the nature of the stenosis. Any one who has seen many patients of this kind will admit that the external habitus is not decisive. The nature of the stenosis can only be determined by a study of the entire condition.

The examination of the diseased organ must be made systematically, and, in every case of gastric disease, should begin *with inspection.* I expressly emphasize this method of examination because in practice it is often neglected. As, in the examination of the heart, the sequence of the investigation is essential, so is it in the stomach. Inspection often gives us important knowledge; naturally, not in very fat persons. On the other hand, in slender persons, poor in fat, with thin flaccid abdominal walls,

we often see the contours of the protruding and dilated stomach without other aid. This, of course, depends upon the *time* of the examination. If we examine immediately after the stomach has rid itself of the greater part of its contents by vomiting or by lavage, inspection usually reveals nothing. If the stomach, however, is still quite full, we can frequently trace its boundaries; the upper portion of the epigastrium, perhaps still somewhat sunken, is seen lower down extending far below the navel, where a protuberance is noted, inside of which there is often active peristaltic movement from left to right. Upon superficial investigation these peristaltic movements may perhaps convey the impression of being irregular; on closer examination, however, a certain regularity is observed, and elevations and depressions may be recognized. The boundaries of the stomach are not only sharply defined, but at the same time we conclude that we are not dealing with a simple flaccidity. In atony such active peristaltic movements are never observed. By inspection alone, we can, under some circumstances, determine the presence of an ectasis, but also simultaneously that the stomach labors with *increased force.* The latter circumstance favors *stenosis*; of course, this need not be an organic obstruction, for even a functional disturbance, a spasmodic contraction of the pylorus, may temporarily make the outflow difficult, and produce an increase of peristalsis.

For a positive diagnosis of dilatation it is absolutely necessary to observe accurately not only the lower but also the *upper* boundary. As is well known, marked dilatations frequently, even as a rule, accompany *gastroptosis,* i. e., the whole stomach is displaced downward. The dislocation of the lower border alone to a point more or less below the navel does not prove dilatation, for this also happens in gastroptosis; therefore, the upper boundary must be accurately located. Normally this is invisible; if it be displaced downward, we note in the epigastrium a slight flattening, and below this a prominence gradually increasing as it descends, and with its convexity directed downward.

I shall not here discuss the results of *electric illumination.* Apart from the fact that this method can only be utilized in isolated instances, it gives no results which may not be obtained in other and simpler ways.

Inspection is to be followed by *palpation.* By this means we confirm the results of inspection and also gain other valuable knowledge. In thin patients, provided the stomach is not empty, we may often distinctly palpate the organ under the flaccid abdominal walls, particularly if the hand is laid flat with the ulnar surface next the abdomen, and, lightly stroking, glides downward. The practised hand frequently traces the boundaries of the stomach accurately. Simultaneously the proof of a tumor in the pyloric region sometimes complements the diagnosis of such cases.

Less importance is to be attached to the method formerly so much practised—of *palpation by means of a sound.* Although it is true that we frequently succeed in feeling the end of the sound through the abdominal

walls, we find at most only the lower boundary of the stomach, and nothing showing its total extension; moreover, a bend in the sound may very readily lead to errors.

Another symptom that may be determined by palpation is the *splashing sound* which we elicit by tapping the abdominal coverings with the finger. Splashing sounds only permit diagnostic conclusions when we *simultaneously consider the time and the extent of their development.* In themselves they only show the presence of fluid and air in a hollow organ. Splashing sounds may, under some circumstances, also be produced in healthy persons, but only when the stomach contains air and fluid, and even when this is the case not beyond the normal boundaries of the stomach. If we find splashing sounds extending beyond these, if we find them within abnormal limits at a time when a healthy stomach should be empty, that is, seven hours after a meal or in the morning before food is taken, diagnostic significance may be attached to them.

Such a splashing sound proves that the stomach still contains more or less profuse amounts of fluid, and it further proves that the stomach is more decidedly dilated than the normal. Naturally, if the latter conclusion is justified, gastroptosis must be excluded.

To determine the boundaries and position of the stomach it is best to *inflate it by means of air or carbonic acid,* and the choice between these two methods has been much discussed. We would digress too far to enumerate all the objections which have been made against inflation by carbonic acid. I believe, however, that these objections are entirely unfounded. I have never seen deleterious effects, or even danger from it, although I have used this method daily for many years. But doses sufficiently large must be given, a heaping teaspoonful of sodium bicarbonate and a correspondingly large dose of tartaric acid; less than this is insufficient.

Distention by means of bellows, and autodistention, such as has lately been advised by Spirack[1] have the disadvantage that a stomach-tube is necessary. The administration of the effervescing mixture is easier and more readily carried out. If the abdominal walls are not too tense and fat we usually succeed, without more ado, in tracing the contours of the stomach; where this is impossible, its boundaries are frequently revealed by the uniformly elastic sensation and by percussion. In stenosis of the pylorus, after the administration of an effervescing mixture, the peristaltic movements of the stomach usually show a marked increase in activity.

Percussion of the stomach serves to confirm and emphasize the previously mentioned results; primarily it only aids us in tracing the boundaries of the stomach, therefore in *ganging its size,* but does not prove a simultaneously existing motor insufficiency. But it does confirm the lat-

[1] *Deutsche med. Wochenschr.*, 1900, Nr. 23.

ter if we succeed in ascertaining the presence of fluid in the stomach at a time when it should be empty. For this purpose it is advisable to examine the patient while in the erect and the recumbent postures alternately. If, when standing, we percuss downward, and if the upper areas of the stomach are first filled, we find a tympanitic zone which is bounded at the height of the navel, or somewhat below or even somewhat higher, by a horizontal dull line, which lower down terminates in a more or less convex curve, but which in the recumbent posture again clears up. To guard against errors, it is advisable in such cases to evacuate the stomach and thus control it. An evacuation of this kind is, *eo ipso,* advisable in all cases where there is the slightest suspicion of disturbance of the motor activity of the stomach.

The Penzoldt-Dehio method, which consists in administering to the patient early in the morning upon an empty stomach two or three glasses of water in succession, and determining after each glass the extent of the gastric dulness while the patient stands erect, is more useful for ascertaining the lower border of the stomach and its tonus than for the proof of dilatation. It may be dispensed with in cases of high-graded ectasis in which the stomach is never entirely empty. Only in mild grades of motor insufficiency is it worthy of trial, and even here it is superfluous since a diagnostic evacuation will give us much more positive proof of the existence of motor insufficiency, and, at the same time, acquaints us with the degree of the same.

Some of the other numerous methods for determining the size and capacity of the stomach are very complicated, have no advantage over those just mentioned, and, for this reason, may be ignored. The same is true of the method recently proposed by Queirolo.[1]

If the methods of examination described have given any evidence of the existence of a dilatation of motor insufficiency of mild degree, we produce a *diagnostic evacuation.* This evacuation, a definite time after a meal, enables us to decide whether or not the stomach has fulfilled its task of propelling the food at the right time into the intestine. As a test of motor activity, the *test-meal* is unquestionably more satisfactory than the test breakfast, for the reason that, in consequence of the greater labor put upon the stomach, we get a better idea of its activity. However, the trial-breakfast may also be utilized to test the motor activity.

A healthy stomach is empty six to seven hours after the intake of a test-meal. If, after this time, remains of food are present, its motor activity is impaired.

The greater the amount of food still present in the stomach, the greater the degree of motor insufficiency. We may, of course, differentiate varying degrees of motor insufficiency according to the amount of the residue

[1] *Verhandlungen des XVIII. Congresses für innere Medicin,* 1900.

and the time it remains in the stomach. Usually it is sufficient to differentiate *two degrees,* in one of which the stomach is not yet empty seven hours after a test-meal but is so in the morning; in the second, a more advanced stage, in which, after a simple evening meal, the stomach is not empty on the following morning.

My usual method is this: In the evening, seven hours after the midday meal, I evacuate the stomach. If there are profuse remains of food motor insufficiency is proven. I then wash out the stomach and permit the patient to take a simple evening meal. The stomach is evacuated the next morning before any food is taken. If it is empty, this shows that the stomach which was unable to digest a test-meal in seven hours is, nevertheless, able to digest a smaller meal, the evening meal, in a longer period —in about twelve hours. If the stomach is not empty upon the succeeding morning, a higher grade of motor insufficiency is evident.

Another method is to wash the stomach clean in the evening, after which the patient is permitted to take the evening meal, and the stomach is evacuated the next morning before any food is taken. The next day, without a previous evening washing, the supper is eaten, and the following morning the stomach is again washed out before any food is taken. In the latter case we will often see, before food is taken, that the stomach still contains remains of food, while, upon the day before, when an evening washing preceded, it contained none. This shows that the evening washing has benefited the condition of the stomach. Naturally this method may be modified in various ways.

Higher grades of motor insufficiency wherein the stomach, even overnight, cannot completely get rid of its contents, as a rule are associated with an increase in size, and this is not surprising. Evidently, the duration of the motor insufficiency has a causative influence in the development of this increase.

In connection with gastric evacuation, there must be an exact examination of the gastric contents. This also occasionally aids us in the diagnosis of motor disturbance, for, very frequently, remains of ingesta which had been consumed days, even weeks, prior, are evacuated,—substances such as cherry-stones, seeds of cranberries, of cucumbers, of pears, currants, etc. Marked gas fermentation also presupposes a prolonged stagnation. The three characteristic layers of the gastric contents (the upper or foamy layer, the medium or fluid layer, the lower or finely distributed starchy sediment) proves stagnation and at least a sufficient HCl secretion. For an exact estimation of slight disturbance in less developed cases Strauss [1] gives valuable advice, which is to take the unfiltered gastric contents, place them in a fermentation tube with grape sugar, and permit them to stand for some time; by this means we get an idea of the rapidity and intensity of

[1] *Zeitschrift für praktische Aerzte*, 1896, Nr. 6.

the gas formation, of the retained causes of fermentation. The further examination of the gastric contents does not aid so much in the exact estimation of motor insufficiency as in the question of the nature and final cause of the dilatation, that is, of the underlying affection. Although by this process only can we understand the nature of the transformation of the ingesta, and from the gastric juice only can draw conclusions regarding the underlying affections which have caused the dilatation and motor insufficiency, nevertheless we obtain from this, particularly when taken in connection with the other symptoms, important points of support. Thus coarse undigested remains of food, particularly of meat, absence of free HCl and profuse amounts of lactic acid primarily indicate carcinoma, while a profuse residue consisting only of fluid and remains of starch with decided yeast fermentation, the presence of large amounts of yeast cells which are beginning to germinate, as well as sarcinæ and an abundant amount of hydrochloric acid, favor benign dilatation.

A constant admixture of bile in the material vomited or evacuated from the stomach points to a deeper seat of the obstruction, to an infrapapillary duodenal stenosis.

In the majority of cases, we may content ourselves with testing the motor activity by simple lavage, and, perhaps, as a control, a subsequent lavage may be performed. If, one hour after a test breakfast, and six to seven hours after a test-meal, a plentiful residue of half a liter and more is found, motor insufficiency is certainly present.

This method is satisfactory in practice. For exact estimation and scientific purposes it is insufficient. Here the method must, at the same time, show us what residue remains in the stomach. Formulas for determining the residue have been given by Mathieu and Remond,[1] Strauss,[2] Goldschmidt,[3] Sörensen and Brandenburg.[4] We cannot enter into the details of these processes. In practice they may all be dispensed with.

Any one who desires to employ still other methods of examination for purposes of control finds a number of such at his service. One is Strauss' currant test, in which the patient is given in the evening a tablespoonful of currants or cranberry jelly, and upon the succeeding morning, the stomach is evacuated after a test breakfast or a test-meal, and an examination is made to determine whether, and how many, seeds are still present in the evacuated contents. In a normal person these are no longer found. Other tests are by the *salol method* and Klemperer's *oil method*. These tests are all inferior to the old process, first employed by Leube, for testing the duration of digestion. The *iodipin* method, lately advised,

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[1] *Soc. de biolog.*, 1890.

[2] *Therapeutische Monatshefte*, 1895.

[3] *Münchener med. Wochenschrift*, 1897, Nr. 13.

[4] *Archiv f. Verdauungskrankheiten*, Bd. III.

which depends upon the fact that the gastric juice is incapable of freeing iodin from the iodin fat, and that the iodin is first set free in the intestine, can, if necessary, be employed as a substitution method, which is not quite free from objection in those cases in which absolutely no use can be made of the stomach-tube. This method has the disadvantage that it consumes more time than lavage, while the latter is superior in that it enables us to form conclusions in regard to the motor activity, and also shows us the secretory condition.

These are the *objective* signs which indicate gastric dilatation and motor insufficiency.

Before we ask what aids we have at command to assist us in determining the *nature* of the dilatation and the motor insufficiency, a few symptoms may be briefly mentioned which are directly connected with the disturbed motor activity of the stomach.

That disturbances of *intestinal activity* are not rarely observed in dilatation of the stomach is not surprising. Patients with this affection frequently have a tendency to constipation; much more rarely they suffer from diarrhea. This constipation may, to a great extent, be due to insufficient absorption of water; the excretion of water from the stomach and the difficult propulsion of the ingesta into the intestine may be the cause of this. In cases of high-graded dilatation the dryness of all the tissues is the reason why more water is withdrawn from the intestinal contents than in the norm. This readily explains the conspicuously hard and dry composition of the feces so often observed, and the difficulty of their propulsion.

The *diminution in the amount of urine* noted in extreme dilatation is due to the decreased absorption of water. The amount of urine voided is a fair measure of the activity of the stomach. In very marked cases, the daily excretion of urine is often decreased to a few cubic centimeters. An increase in the amount of urine is an infallible sign of improvement; the slighter the amount, the more concentrated it naturally is, and the higher its specific gravity. In dilatation with a plentiful HCl production, the urine often shows a neutral or alkaline reaction, and also when, in consequence of profuse vomiting or by frequently repeated lavage, much acid gastric juice is removed.

Occasionally we note an action upon the *heart* shown by a *slowing of the beat*. However, this occurs not only in dilatation, but also in many other gastric affections, and, therefore, may in nowise be designated a pathognomonic peculiarity of dilatation.

More important than the previously mentioned effects are certain derangements of the *nervous system*. Among these the symptom-complex designated as *tetany* is especially interesting. This, as is well known, manifests itself particularly by tonic muscular spasms in the hands and arms, occurring intermittently, and an increased irritability of the nerves.

Fortunately, tetany is not among the frequent sequels of dilatation. Most cases of tetany in gastric dilatation observed up to the present have occurred in benign pyloric stenoses, mostly in such as were accompanied by hyperacidity and acid fermentation; however, cases of tetany have also been observed in carcinomatous pyloric stenosis, in absence of HCl secretion, and in lactic acid fermentation. How tetany is produced in these cases is still a mooted question. With Kussmaul, some attribute it to a deficiency of water in the organism; in another theory, the development of the attacks of spasm is attributed to a reflex irritation of the nerve center from the irritated nerves of the gastric mucous membrane; in a third theory, gastric tetany is regarded as the consequence of an intoxication of the organism by abnormal products of metabolism in the diseased gastrointestinal canal. So far as I am able to judge, the last theory has found the most adherents. A discussion of these various theories would lead us too far from our subject; and, naturally, I must decline to enter upon a special description of the symptoms of tetany. (See volume "Diseases of the Nervous System.")

Headache and vertigo are not rarely the sequels and accompanying symptoms of dilatation; usually these are not very intense. In the terminal stages of marked dilatation the patients are sometimes observed to become soporose, and finally succumb to coma. Such a case, in which sopor and general spasms occurred and terminated fatally after a few days, has recently been reported by Jürgensen.[1] In this case the fact was noteworthy that during the soporose condition the secretion of urine ceased entirely. The supposition of an autointoxication in such cases is certainly very reasonable.

The appearance of curious *drumstick-like changes in the fingers and toes* in a case of gastrectasis due to benign pyloric stenosis must be mentioned. This case, recently reported by Dennig,[2] is interesting for the reason that after a successful operation (pyloric resection) there was a complete retardation of the drumstick-like changes.

If, after an examination by the methods detailed, gastrectasis and motor insufficiency have been determined, we then inquire what has produced the condition, whether a mechanical obstruction at, or in the neighborhood of, the pylorus, or simple flaccidity. The question may sometimes be decided at a glance, or immediately upon palpating the abdomen, in other cases only after minute consideration of all the symptoms and after prolonged observation. If the condition were as simple as has recently been maintained, that every chronic dilatation is due to a hindrance in passage or to a local process at the outlet of the stomach, the diagnosis of ectasis, and also of "pyloric stenosis" could be at once made. But the conditions are not so simple, although it is true that the most marked

[1] *Deutsches Archiv f. klin. Medicin*, Bd. LX.

[2] *Münchener med. Wochenschrift*, 1901, Nr. 10.

grades of dilatation occur in pylorie stenosis. In practiee, however, we must diagnostieate not only the marked grades of ectasis, but also the milder grades, the earlier stages.

Such forms are due to an atonic condition. But spastic proccsses also, without an organic stenosis being present, occasionally, as we have before stated, play a rôle in the etiology of dilatation.

Atony, as we have seen, oecurs in persons whose nutrition has suffered, and in those with flaecid abdominal walls; not rarely, gastroptosis and enteroptosis exist simultaneously. Sueh an atonic stomach, as a rule, has a flaccid feeling with but little tension. In spite of a sufficient secretion of gastrie juice, as shown by lavage, we frequently find eoarse particles of food, especially fibers of meat. The latter are probably due to a loss of energy of the muscularis whieh prevents an intimate admixture of the ingesta with the gastric juiee.

A successful treatment, in such eases, often confirms the diagnosis. If, by suitable diet and by improving the nutrition,—briefly, by methods which strengthen the tonus of the muscularis and improve the general nutrition,—we can remove the symptoms of motor insufficieney, this result favors the view that we are not dealing with an organie stenosis.

DIFFERENTIAL DIAGNOSIS

In the *differential diagnosis between atonic and mechanical dilatation,* the history is important. When symptoms of ulcer have previously existed, when in connection with this the pieture of dilatation has gradually developed, our first thought is that a eieatrix of the pylorus has produeed stenosis. If examination reveal a uniform resistance in the pyloric region, if the seeretion of gastric juiee be normal or increased, if we observe active peristaltic movements, a cieatrix from ulcer is indicated. When, upon inflation, we find that the thiekened area remains immobile at the same place in which it appeared while the stomach was empty, this we assume to indicate a simultaneous perigastric adhesion.

The diagnosis is more diffieult in cases in which *hypersecretion and dilatation* exist simultaneously. I have already discussed the varying possibilities of this combination. Aetive peristalsis favors a mechanical obstruction, although not neeessarily an organie hindranee. Preceding gastric hemorrhage favors ulcer, and, possibly, a cicatricial stenosis, although this is by no means absolutely proven. The results of therapy often indicate that the eause which brought about the dilatation eould not well have been an organic stenosis.

Probably the most positive sign of meehanieal obstruction at the pylorus is *active peristalsis.* It is never absent except in the terminal stages and in those instances in whieh secondary degeneration of the hypertrophie musculature has oeeurred. Yet the obstruction is not neeessarily an organic

one. As is well known, spasms of the pylorus play a part in many forms of disease of the stomach as well as in dilatation, and are particularly prominent in those which run their course with increased secretion of gastric juice. According to Serejukow, acids cause the sphincter muscle to contract.

A factor not without significance in the differential diagnosis between these forms of dilatation is the *rapidity of the influx and outflow* of the gastric contents; that is, of the water used for lavage. The stronger the tonus of the stomach, the more powerful the musculature in an obstruction at the outlet of the stomach, the greater the force with which the organ will empty itself. The more slowly the water used for lavage flows in, the more rapidly will it flow out. The conditions are the reverse of this with very flaccid, thin, atrophic gastric walls. Here the gastric contents are discharged very slowly and incompletely; the water poured in, however, rushes with force through the tube, so that swirls develop, and air is readily aspirated into the stomach.

An admixture of *bile* is not infrequently observed at the termination of a washing of the stomach, especially in atonic dilatation. If other signs of stenosis exist, the permanent regurgitation of bile favors an infrapapillary seat of the stricture.

The most important sign of stenosis, however, is always the *demonstration of a tumor* at, or in the vicinity of, the pylorus. The tumor, in itself, does not prove a stenosis, for we must be certain that it belongs to the stomach or to the adjacent portion of the duodenum.

When a tumor has been diagnosticated, the question to be next decided is whether it be benign or malignant. We would digress too far if we entered upon this differential diagnosis. This point can only be established by considering the duration of the disease, the condition of the gastric contents, the secretion of the gastric juice and the like.

That the *results of treatment* may be utilized to a certain extent in diagnosis, particularly in differential diagnosis, whether stenosis, or atony, or only functional spasm be present, is clear. Rapid relief of the dilatation by suitable treatment favors the atonic form or spastic stenosis. Naturally, in a temporary improvement, we should not at once permit ourselves to make this diagnosis. Ectases due to organic stenosis are also ameliorated by methodical lavage, by diet, and other remedies; but this improvement is not permanent.

COURSE OF THE DISEASE

Having described the symptoms of motor insufficiency and dilatation, it is next expedient to describe their course. In the majority of cases the clinical picture of dilatation develops gradually; rarely does it happen that a single deleterious effect, brief in duration, suddenly causes dilatation.

In the course, as in the development, we must differentiate between *acute* and *chronic dilatation*. The acute varieties, as a rule, are rapidly cured by appropriate treatment; yet, repeatedly, cases with a fatal termination have been observed. In explanation of these, Albrecht's previously mentioned investigation of arterio-mesenteric intestinal occlusion at the duodeno-jejunal boundary might well be considered.

Whether, in all cases of acute gastric dilatation running a fatal course, mechanical factors of this kind play an important rôle, cannot, naturally, be subsequently decided. The symptoms of severe acute dilatation have several times appeared without any demonstrable cause. In a few cases the clinical picture developed directly after anesthesia; in several cases a severe infectious disease immediately preceded the condition. It is worthy of note that in a number of these cases vomiting was absent during the entire course; this is probably to be explained by the high-graded, over-distention and loss of tonicity of the walls of the stomach.

Such severe, rapidly fatal cases are very rare. Much more frequently mild and moderately severe forms are observed, the origin of which is traced to gross errors in diet. A marked, painful swelling of the abdomen soon occurs and is associated with nausea, vomiting, and great thirst. In mild cases the symptoms disappear in a few days, particularly if thorough lavage is performed, and we must be careful to see that the stomach is absolutely emptied. In other cases the symptoms do not yield so readily. The earlier and more energetic the treatment, the more favorable and rapid, as a rule, is the course.

In private practice, we more frequently deal with *chronic* cases—dilatation which develops gradually. The course of these varies greatly, and depends upon different factors: the underlying cause, the mode of life, and numerous other conditions. Not infrequently we notice a change in the symptoms, sometimes amelioration, at other times aggravation; this is observed, above all, in cases of non-organic stenosis. Organic stenosis, on the other hand, generally shows a progressive increase in the symptoms. The most marked degrees of dilatation are met with in pyloric stenosis; atonic dilatation, in the majority of cases, is extremely slight. Yet it appears to me that we are not justified in denying absolutely the occurrence of atonic dilatation, as some writers do. At all events, it is rare, and usually not of marked extent.

It is unfortunate that the test by physicians of the motor activity has not everywhere been regarded as of equal value with the test of the secretory activity. Nevertheless, we must admit that the disturbances of motor activity are often of the greatest importance, even more so than the variations in the secretion of the gastric juice. If in every serious gastric disturbance the same importance were attached to the motor as to the secretory activity, many a case which is now recorded under the name "chronic catarrh" or "nervous dyspepsia" would be recognized as motor

insufficiency or as dilatation, although not of extreme degree. I have met with a considerable number of such moderate dilatations which, under appropriate treatment, proper diet, rest, forced feeding, methodical lavage and the like, were entirely cured. These patients with atony, even after relatively light meals, have a sensation of satiety, of pressure or fulness in the stomach; they feel indisposed to follow their occupations; they are listless. As a rule there is no complaint of actual pain. If gastroptosis goes hand in hand with atony, as it frequently does, the disturbances are increased. The patients, after eating only a little food, have very disagreeable sensations, they lose their appetite, and gradually the nutrition suffers. Such atonies are very often disregarded, or erroneously diagnosticated, and if they do not early come under suitable treatment they rapidly increase in severity. Only a minute objective examination and the aid of an accurate history make an early diagnosis possible. The more promptly suitable treatment is instituted, the easier is the cure. It can be readily understood that these forms show frequent changes, a tendency to relapse, and that periods of amelioration and aggravation alternate.

A much more uniform course is generally observed in motor insufficiency and dilatation due to organic stenosis. Here, also, transitory improvement may occur; but it cannot last long unless we succeed in removing the obstruction which causes the stenosis; however, this is hardly possible except by surgical interference. We cannot enter into details of the modifications of these forms on account of the great variety of their causes.

DIAGNOSIS

In discussing the diagnosis of motor insufficiency in dilatation, I may be brief. This is based solely upon the *objective* examination, *upon the proof of prolonged digestion, and upon an increase in the size of the stomach.* Among the methods that have been mentioned, the most certain one for determining motor insufficiency is to wash out the stomach six to seven hours after a test-meal, or two hours after a test breakfast. Splashing sounds heard at a period in which the stomach should be empty assist the diagnosis, but never render the diagnostic washing out unnecessary. The latter has the advantage that it simultaneously enables us to form conclusions in regard to the secretory activity, and also to determine what processes cause the dilatation.

The *degree of motor insufficiency* is readily estimated from the amount of retained material and the period of its retention in the stomach.

Considering the diagnostic criteria which have been mentioned, confusion with other affections is hardly possible. *Megalogastria* could only be mistaken for dilatation if we should neglect the washing out of the stomach which is absolutely necessary to test the motor activity. Still less would it be possible to confound the condition with chronic gastritis

or with nervous dyspepsia. This confusion is impossible if diagnostic lavage be performed. However, atony and motor insufficiency may occur simultaneously with chronic gastritis and nervous dyspepsia; motor insufficiency is only a disturbance of function which may develop in various ways.

In practice confusion of gastrectasis with *gastroptosis* occurs. This error is not rare, for the reason that with a low position of the greater curvature of the stomach and a succussion sound developed below the navel dilatation is assumed without further investigation. This is not justified by the facts. The proof of displacement of the lower boundary of the stomach is not sufficient to determine dilatation; its total extension must be determined, not only that of the lower, but also of the upper and the lateral borders. The investigation of the latter is interesting. As Michaelis [1] has shown in cases of enlargement of the stomach in which the gastric motility was severely disturbed, the right border, as a rule, is found much farther removed from the median line than in a stomach with normal motility.

On the other hand, we must remember that motor insufficiency and dilatation are often combined with gastroptosis. It is quite natural that a permanent over-weighting of the stomach should lead to a sinking, to gastroptosis. In fact, in almost all extreme dilatations, we find more or less sinking of the lesser curvature. An extreme case of decidedly low position of the stomach which may readily be recognized (with a high grade of dilatation) may be seen in the accompanying illustration (Fig. 10) in the case of a woman, aged 59, who had carcinomatous stenosis of the pylorus.

Simple atonic dilatations very frequently are combined with gastroptosis. This combination is not difficult to recognize, provided we make use of the previously mentioned diagnostic aids.

By what means atonic dilatation is to be distinguished from that caused by mechanical obstruction has been stated. This question can often only be decided after prolonged observation, under some circumstances only by studying the results of treatment.

Much more difficult is it to decide as to the nature of the constriction. Whether we are dealing with *benign* or *malignant* stenosis is a most important question, and, in the majority of cases, not difficult to decide. A hard, nodulated tumor in the pyloric region, abundant coarse residue with frequent hemorrhagic admixture in the form of coffee-ground masses, absence of free HCl, lactic acid fermentation, and rapidly increasing cachexia, naturally favor malignant stenosis. But, in malignant stenosis, particularly in such as develops from an ulcer, we often find a normal secretion of gastric juice. It must be borne in mind that *no single symptom is decisive*, but the combination of all the symptoms is necessary to a diag-

[1] *Zeitschr. f. klin. Med.* XXXIV.

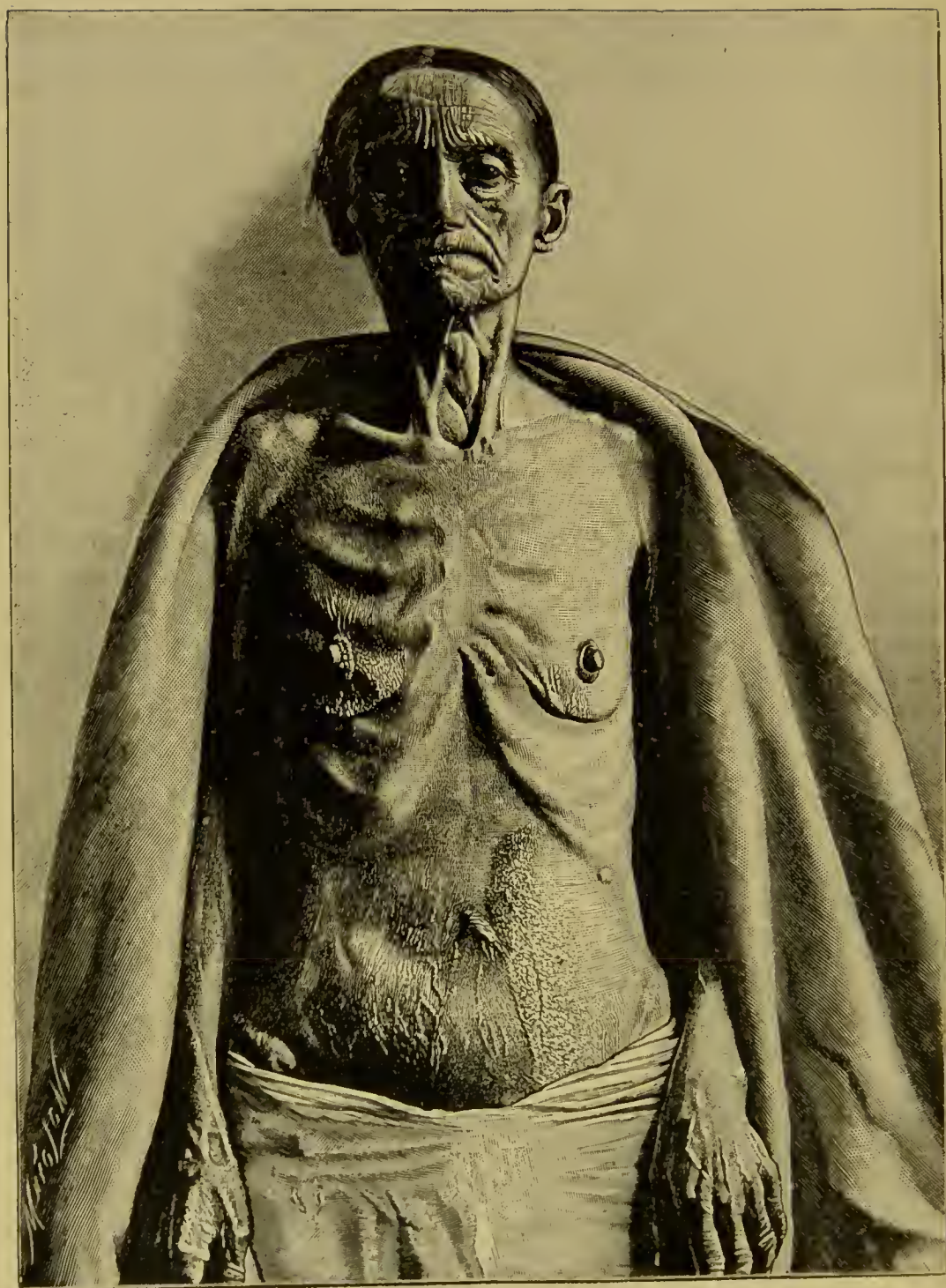

Fig. 10.

nosis; whether one or the other is absent is not conclusive. But a diagnosis can never be looked upon as certain which does not combine all the symptoms.

PROGNOSIS

As has been shown, the importance of motor insufficiency and dilatation varies greatly in the individual case; it may run an *acute* or a *chronic* course. At one time it represents a transitory atonic condition, at another time it depends upon a mechanical obstruction, which in one case may be benign, and in the other malignant. The prognosis of motor insufficiency in dilatation, therefore, also varies strikingly in the individual case. Some forms are cured in the briefest time by proper management, others only after months or even years of treatment; some in the briefest time terminate fatally, others again are not susceptible to internal treatment, and surgical measures alone will cure the affection or moderate its consequences.

The earlier treatment is begun, and the more systematically it is carried out, the more rapidly do *acute* dilatations improve. Under some circumstances threatening symptoms may intervene, and the cases even terminate fatally. Generally no prognosis is possible in *chronic* dilatation. Many factors influence the prognosis in a given case; thus, the duration of the affection and the degree of motor insufficiency. Moderate degrees of motor insufficiency which have not existed for a long time naturally lead to a more favorable prognosis than long-existing, high-graded dilatations. But, above all, the prognosis depends upon the underlying *cause* of the dilatation. If we succeed in removing the causative factor, as a rule we also succeed in ameliorating the dilatation. Mild and medium grades of atony under suitable treatment are often cured in the briefest time. Where the cause of the dilatation is an organic obstruction in the pylorus or its vicinity, improvement can only be brought about by surgical means. Internal treatment in such cases produces apparent, but in reality transitory, results. Unfortunately many cases are no longer suitable for operative interference, such as extensive carcinomata, or those in which metastases are present and the like. The dilatation alone does not permit a positive prognosis, but this depends, above all, upon the underlying affection; that is, upon the cause which has produced the dilatation.

TREATMENT

We now come to the main question: *How shall we treat dilatation?* Naturally it is not my object to explain here all the methods of treatment and the other procedures which may be necessary on account of the causative factor, carcinoma, cicatrix from ulcer, perigastric adhesions, and similar conditions; we should digress too far from our actual theme. But

we shall consider only the treatment of motor insufficiency and dilatation as such.

Acute dilatations which have been brought about by over-loading the stomach with food difficult to digest require immediate and thorough emptying of the stomach and an absolute rest of the organ for some time. When debility necessitates the administration of food or fluid, rectal alimentation should be resorted to. Under all circumstances, however, the stomach must have absolute rest in the days succeeding. How long this treatment is to be pursued naturally depends upon the severity of the symptom-complex. In acute dilatation in which symptoms of obstruction appear, it is not enough merely to empty the over-filled stomach, and to suspend for some time the administration of food and fluid by the mouth; we must also attempt to relieve the coils of the small intestines at their mesenteric roots, which, by a drawing process, have been forced down into the pelvis. For this purpose, the patient must be placed in such a position that the small intestine may find its way out of the pelvis, and the tension of its mesentery be quickly relieved. Therefore the knee-elbow position, or lying prone upon the stomach, is most effective. Cases of this kind ending in recovery have recently been reported by Müller.[1] Where this method does not bring relief, eventually surgical measures only can avail.

The conditions are different in the much more frequent *chronic* forms of dilatation and motor insufficiency with which the physician must chiefly concern himself. Although our object varies according to whether we are dealing with simple atony, with atonic dilatation, or with dilatation caused by a mechanical obstruction, nevertheless, certain therapeutic rules are equally operative in all these forms. In these groups, prolonged stagnation, and all abnormal weighting of the stomach, must be avoided as much as possible, that is, combated, and the diet must be so ordered that it will necessitate the least possible exercise of the motor activity of the stomach, and can by no possibility be injurious.

Our primary object in dilatation is to regulate the diet. We must invariably consider first, the kind of food, and, secondly, much more important in dilatation, the form in which it is administered. In modern practice the majority of physicians advise a dry diet in dilatation; fluids are to be refrained from as much as possible, for they load the stomach by their weight, are badly absorbed, and, in pyloric stenosis are expelled from the stomach with difficulty as is proven by the over-distention of the stomach with fluid. A dry diet, therefore, with the greatest possible limitation of fluids, is advisable.

In every form of dilatation, whether it depend upon atony or upon mechanical obstruction, the motor activity of the stomach is insufficient, either absolutely or relatively insufficient, according to the amount of labor

[1] *Deutsche Zeitschrift für Chirurgie*, Bd. LVI.

required of it. We must reckon with this motor weakness; i. e., we must choose a diet which necessitates but slight exertion on the part of the motor power, which does not require over-exertion, but one that prevents it. For, by means of the motor power, the fragments of ingesta are split up in the stomach, reduced in size, liquefied, and, finally, propelled from the stomach into the intestines.

v. Mering, by his well known researches, has proven that the healthy stomach possesses only very slight power of absorption, and that its labor consists chiefly in propelling the fluid gastric contents into the intestines. Moritz, too, has demonstrated by investigations that fluid food is more rapidly propelled into the intestine than pappy food, and this again in a shorter time than solids.

Therefore, above all, in motor insufficiency and dilatation we must choose a diet which is readily forced onward into the intestine. This is true, however, only of fluid and pappy food.

In opposition to the almost universal practice of employing a dry diet, I have for years advised *fluid* and *pappy forms of food in dilatation.* This method appears to have recently gained many adherents. In the choice of the diet we must always reckon with the weakened motor power, and, therefore, so far as possible, choose a food that may be broken up into fine particles.

One objection has been raised to fluid diet, namely, that the nutritive value of fluid food, in proportion to its volume, is but slight. This argument may, however, be met by choosing a food as nutritious as possible. Such a nutriment is represented by *milk,* which may be given pure, or as buttermilk, as cream, as milk-custard, and as gruel with various additions of grits, rice, and the like. The taste may be varied by the addition of aromatic substances—by brandy, lime-water, by the addition of cocoa, vanilla, cinnamon, lemon, etc. We can generally succeed, in almost all patients, in administering larger or smaller amounts of milk, even when they express a dislike for it.

Eggs may be variously prepared and, in addition to other foods, take a place in the diet of the patient. The addition of tropon, nutrose, plasmon and similar preparations to fluid and pappy food increases their nutritive value.

Meat, in so far as the underlying affection permits its use, had best be given scraped or chopped. Calves' brains and sweetbreads, on account of their pulpy consistence, are very suitable for such patients. Game must be avoided, as well as goose, duck, and fat pork. Of fish the most suitable are giant pike, pike, perch, and shell fish. Vegetables are to be given only very finely split up or in the form of purée,—of asparagus the heads, of cauliflower the tops, and mashed potatoes.

Fat, especially milk-fat, is also suitable for patients with disturbances of the gastric motility, particularly for such forms as are combined with

hyperacidity. Formerly the opinion prevailed that large amounts of fat lie heavy in the stomach [1] and that in disturbances of its motility they should be avoided. The researches of Strauss,[2] Bachmann,[3] Wolkowitsch [4] and others, show that too much cream will diminish the secretory power of the stomach, and even in the stomach showing motor insufficiency it is subjectively better borne than a large quantity of carbohydrates. In hypersecretion, and particularly when disturbance of motility is simultaneously present, the profuse ingestion of undissolved carbohydrates increases hypersecretion. For these cases of disturbance of motility with well maintained HCl secretion, it, therefore, seems more rational to substitute for the carbohydrates as far as possible the fats which are readily and thoroughly broken up. Here the *albumin fat diet* for several weeks is advisable.

It is well known that some patients cannot digest fat, particularly cream. In these cases of motor insufficiency and dilatation, the opinion previously maintained that fat must be prohibited and that, at most, but small quantities of butter should be permitted, is no longer in accordance with our clinical experience.

A definite *diet list* for patients with dilatation cannot be given; it is clear that wealthy patients may have a more varied dietary than the poor, but for both the rules given above, which primarily enjoin a rest for the stomach, are alike operative.

Another self-evident rule is that such patients should *never take too much food at one time.* The custom of eating only two meals a day is not suitable for patients with gastrectasis. The meals are to be small, but they should be frequent. Under some circumstances, the method of Albu [5] of giving the patient fluid, concentrated food through the stomach-tube immediately after the morning and evening stomach washing may be adopted; but this would come into consideration only in the nutrition of very debilitated patients with marked anorexia.

In very emaciated persons, to improve the nutrition and to rest the stomach as much as possible, a portion of the fluid food may be given by the rectum. If we are only endeavoring to combat the decrease of water in the organism, enemata of water with the addition of salt (1 teaspoonful of table salt to a liter of water) or a small quantity of brandy, or wine bouillon enemata ($\frac{2}{3}$ bouillon and $\frac{1}{3}$ Rhine wine) may be given. It is advisable to give nutritive substances admixed with the enema. For this

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[1] In regard to the question of the part played by the stomach in the digestion of fats, I refer to the investigations of Volhard upon "Absorption and Fat Splitting in the Stomach," recently published from my Clinic. (*Münchener med. Wochenschrift*, 1900, Nr. 5 u. 6, und *Zeitschr. f. klin. Med.*, XLII.)

[2] *Zeitschrift für diätetische und physikalische Therapie*, III.

[3] *Archiv für Verdauungskrankheiten*, V.

[4] *Wratsch*, 1898, Nr. 13, in *Archiv für Verdauungskrankheiten.*

[5] *Deutsche med. Wochenschr.*, 1900, Nr. 11.

purpose a peptone-enema (50 to 60 of peptone to 300 of milk), milk and egg enemata (3 eggs with the addition of 3 grams of table salt, in 300 of milk) and starch enemata (50 starch to 300 of milk) may be employed. Solutions of sugar are liable to produce irritation of the mucous membrane of the rectum. If they must be employed, not more than 15 or 20 grams of sugar to 300 of fluid should be used. Nutritive enemata of this kind must of course only be given in extreme dilatation, and for a short time; as complete substitutes for the administration of food by the mouth, they naturally fail. Subcutaneous injections of fat may, under some circumstances, be given for nutritive purposes.

Alcohol is generally to be avoided in dilatation. It is true the stomach will absorb alcohol, but, with its absorption, a plentiful excretion of water from the stomach occurs, which is the greater the larger the amount of alcohol absorbed.

Although these diet rules apply in general to every form of dilatation, whether it run its course with normal or defective secretion of gastric juice, nevertheless, this secretion of gastric juice should be considered in the choice of food. Where it is more or less decreased, milk foods, strong soups, and the most tender meats are indicated, the meat to be finely chopped or divided. But in hypersecretion and hyperacidity, the carbohydrates must be limited, and a meat-fat diet is preferable. In cases of this kind where the secretion of gastric juice is due to pyloric stenosis, where, therefore, notwithstanding dilatation, gastric peristalsis is still active, it may be expected that the stomach is still able to digest meat divided less finely. At all events we need not be so careful in this respect as in the cases with diminished secretory power and simultaneous atony.

As with every damaged or weakened organ, so in the case of the stomach it must be our first object to give it rest. The diet should be light, but, nevertheless, sufficient food must be taken. Rest of the organ is facilitated if the patient with dilatation is kept as much as possible in a recumbent posture.

That an upright position of the body is more unfavorable for the dilated stomach than a recumbent one is clear. After eating, the patient should lie down for a long time. Lying upon the right side after eating has frequently been recommended to facilitate the propulsion of the food mass into the duodenum. It is self-evident that such patients must *eat slowly and masticate thoroughly.*

If the dilatation and the motor insufficiency are due to the difficulty of expelling the gastric contents, we must resort to measures that will assist in this expulsion.

For this purpose systematic massage has been advised. Massage of the stomach, however, also serves the further purpose of strengthening the gastric musculature. If we intend the massage merely to produce a more rapid propulsion of the ingesta into the intestine, it should not be given

immediately after a meal but a considerable time later. Massage is contraindicated in cases of decided fermentation, which must first be removed. In the majority of cases the results of massage are not very satisfactory. We cannot describe the technic. Massage of the stomach, however, should always be given by the physician.

In atony the *faradic current* is useful to strengthen the muscularis of the stomach; this, as a rule, is employed extraventricularly, more rarely intraventricularly. Certain *hydropathic procedures,* douches, the fan-douche, and, particularly, the so-called Scotch douche, are beneficial. From my experience I cannot advise internal douches; in extreme dilatation I do not consider them free from danger, and in milder forms more simple methods may be substituted.

To promote more rapid propulsion of the gastric contents into the intestine in cases of organic or spastic stenosis of the pylorus, the use of olive oil has recently been recommended. Cohnheim [1] advises 50 grams one hour before meals three times daily. Not only in cases of spasm of the pylorus, but also in cicatricial stenoses of the same, he claims to have had good results. In several of these cases, there was scarcely a hope of recovery except by surgical interference.

All of these methods are designed *to rest and strengthen* the stomach; they may suffice in mild grades of motor insufficiency and atony, but not in the severe forms of dilatation in which the stomach is never perfectly empty, but even in the morning, before taking food, portions of that consumed the previous day are still present. Here it is necessary to relieve the stomach of its abnormal contents by *gastric lavage.* In all severe forms of motor insufficiency and dilatation, *gastric lavage* is an extremely valuable and indispensable aid.

The degree of motor insufficiency which necessitates methodic lavage is a disputed point. All authors are unanimous that, in the extreme forms in which the stomach in the morning before breakfast contains food from the day previous, it should be washed daily. It is different in slighter dilatation. In my opinion, even mild forms of dilatation indicate regular lavage. The normal stomach acts only periodically, not continuously. Six to seven hours after the midday meal it is empty, and four to five hours after a simple evening meal it should no longer contain food. In consequence of this, the stomach has certain periods of rest. The diseased stomach with motor insufficiency should have no greater work, and no longer hours of labor, than correspond to the norm. If the stomach in the evening, before supper, still contains larger or smaller quantities of food, it must be washed out. If this is not done it is abnormally taxed, and if the debilitated and distended stomach with motor insufficiency is not to be still further stretched and taxed, naturally no more labor must

[1] *Archiv für Verdauungskrankheiten,* Bd. V.

be exacted from it than from a normal organ. This would, however, be the case, if we expect an evening meal to be digested besides the remains of the midday meal which is still in part undergoing fermentation. This would not ensure to the diseased organ the rest which is of such primary importance.

If the stomach that has been washed before supper is empty upon the following morning, this *evening lavage alone* will be sufficient; if not, it must be performed in the morning in addition. This is not only necessary to relieve the stomach, but because abnormal fermentation and decomposition occur upon prolonged stagnation of the ingesta.

Many recommend lavage in the morning before breakfast. They believe that if the stomach is not empty at this time it should be washed out, and that this is the best time for the procedure. They also believe that at this hour the washing can be most rapidly performed, and is the least exhausting. I do not coincide with this reasoning. Lavage should relieve the stomach by removing its decomposed products of abnormal fermentation.

It is undeniably true that lavage is most easily performed in the morning, and more rapidly because the stomach before breakfast contains relatively the slightest residue of food. But this method gives little relief to the stomach; it has no periods of rest as in the norm. It is better, as others advise, to carry out the lavage late in the evening between 9 and 10 o'clock. This gives absolute relief and an actual period of rest follows. To me it appears most rational to use lavage immediately before the evening meal. If, after seven hours, much residue is still present, which must, under any circumstances, have begun to ferment, it is wiser to remove this and to introduce fresh food into the cleansed stomach than, as the exponents of lavage in the late evening recommend, to add an evening meal to the fermenting remains and evacuate the stomach two or three hours later. If, after evening lavage, the stomach is not empty upon the following morning, it must, in addition, be washed in the morning.

Therapeutic washings should be as thorough as possible, not only to empty the stomach, but to remove as well the causes of fermentation. This should be done first with lukewarm water; medications may be necessary in subsequent washings on account of special individual conditions, because of acid, abnormal fermentation, and the like. The latter, especially, often renders the use of drugs necessary, such as salicylic acid (1–1000), boric acid, resorcin, and similar antiferments.

A thorough cleansing of the stomach is more rapid when there is relatively increased gastric tonus, such as we meet with in pyloric stenosis rather than in the atonic forms. Naturally, lavage must be continued until the stomach is thoroughly cleansed, or until we have convinced ourselves of the uselessness of further effort.

To the *physical aids* which play but an indirect rôle in the therapy of dilatation, *bandages* may be added. They do not reduce the dilatation, but they give the dilated stomach a firm support; they not only assist in producing subjective amelioration, but to a certain extent they relieve the condition. They are particularly valuable in those numerous cases in which, besides dilatation, there is also gastroptosis, or where there is, at the same time, a marked degree of flaccidity of the abdominal walls.

These are the chief remedies and methods to which we resort in dilatation of the stomach. Actual *drug medication* for dilatation does not come into question. It has been maintained of some remedies, such as *strychnin*, that they stimulate the tonus and peristalsis of the stomach, which has also been claimed for *creosote, orexin* and other drugs. But I have observed no remarkable action of this kind from any of the remedies applied.

A few words must be devoted to the *belladonna preparations* and to *atropin* since these remedies have, to a high degree, an antispasmodic effect. Spasm of the pylorus, however, often exerts an influence in dilatation, particularly in those varieties running their course with hyperacidity and hypersecretion. As I have shown, the belladonna preparations not alone inhibit the secretion of gastric juice, but are, at the same time, antispasmodic. On account of these properties, they may be employed with advantage in the two forms of gastrectasis even though only as symptomatic remedies.

The *alkalies* also, even if only indirectly, are useful in certain forms of dilatation, especially in those varieties in which there is hypersecretion. Here they prevent the abnormal production of acids, and assist in the digestion of starches as well as the propulsion of the gastric contents.

I cannot enter minutely into an enumeration of drugs for the relief of *individual symptoms*; they do not affect the dilatation as such, but only certain symptoms caused by the underlying condition.

Tetany, a symptom-complex closely related to ectasis, shall be briefly discussed. Views regarding the origin of tetany and gastrectasis are diverse. The majority of authors adhere to the intoxication theory, and regard tetany as the consequence of an intoxication of the organism with abnormal products of metabolism from the diseased gastrointestinal canal. If this theory be correct, the therapeutic indication is that prolonged stagnation must be prevented. The active treatment of stagnation is, accordingly, the prophylaxis of tetany. Remedies which relieve attacks of tetany, or prevent its occurrence, are unknown.

Unfortunately, the number of cases of dilatation in which the remedies just mentioned do not bring relief is very great, and in these the only hope of improvement or cure is in *surgical measures*. These may serve two purposes, either in removing the actual cause of the dilatation,

or in some manner bringing about the compensation of the damaged motor power.

By far the most frequent indication for surgical interference is seen in *pyloric stenosis*, immaterial whether the stenosis be due to a cicatricial narrowing, to a carcinoma, to compression from without or elsewhere. Where it is possible entirely to remove the stenotic obstruction, this should always be the primary aim.

When the surgical operation is decided upon, the proper time for it should be determined by a full consideration of all the conditions. Malignant stenosis should be operated upon as early as possible. Radical removal of a carcinoma is most desirable. Benign stenosis also necessitates surgical interference, but here we must usually wait until the uselessness of internal remedies has been proven. Where the internal clinician cannot ameliorate the condition with the remedies at his command, where, in spite of all the described methods it is impossible to introduce sufficient nourishment into the organism, the surgeon's art must be called into play.

If the radical removal of the stenosed area be impossible, prevention of the stenosis and compensation may be brought about by the formation of new outlets. *Gastroenterostomy* answers this purpose.

There are, however, also *relative indications* which are the result of special circumstances. Thus, we occasionally see patients whose condition is endurable so long as they remain in a hospital where proper diet and other auxiliary remedies are at hand to maintain them, and where they are able to help themselves. If they return to their former unfavorable surroundings, are compelled to work hard, and are unable to spare themselves, aggravation at once occurs. Here, the unfavorable circumstances of life, not the severity of the disease, compel operative interference which, under other circumstances, might have been postponed. Without an operation such patients as these would succumb in a very brief time. Here the operation is not absolutely indicated, but relatively, on account of the special environment of the case in question.

What operation (resection, pyloroplasty, gastroenterostomy, digital divulsion of the pylorus, etc.) is to be performed in the given case, can only be determined after opening the abdominal cavity. Radical removal of the obstruction which causes stenosis is primarily the aim, even though this very frequently cannot be attained.

Atonic dilatation also may, under some circumstances, although much more rarely, require surgical relief; this does not affect the atony, as such, but serves to facilitate the outflow.

Gastroplication cannot restore the tonus, but only decreases the volume of the organ, and in atony with this alone very little is attained. On the other hand, gastroenterostomy, while it does not remove the atony, prevents stasis, and under some circumstances restores the tonus of the organ. But, with the early recognition and proper treatment of atony,

as a rule it rarely attains such a high degree of development that surgical intervention becomes necessary.

It is not our object to enumerate the individual surgical operations; the internal clinician must perceive the indications for surgery; the choice of the method must be left to the surgeon.

GASTRIC ULCER AND GASTRIC HEMORRHAGE

By C. A. EWALD, Berlin

ETIOLOGY

In no other form of gastric disease is our insight into the finer pathologic processes and the methods which must be adopted for their cure so clear as in *gastric ulcer, round ulcer of the stomach,* or *peptic ulcer,* as the affection has been designated according to its peculiar features.

My actual experience regarding this disease cannot be questioned when I state that, in the last twelve years, I have seen and treated 1250 cases either in the Hospital, the Clinic, or in my private Clinic (not including my office practice). At this point, however, I do not intend to give a text-book description, but I shall consider chiefly the clinical aspect of the pathologic picture, and shall limit myself to the experiences of the last few years.

We differentiate the *acute* and *chronic* forms of gastric ulcer, not, however, by the different nature of the process, but by the rapid or slower course, and under this the various symptoms will be included.

Since Cruveilhier first gave us such a minute and comprehensive description of gastric ulcer (which had formerly been looked upon as a post mortem curiosity) that it constitutes a well-defined and easily recognized pathologic picture, clinical observers and experimental investigators have vied with each other in eagerly studying this affection.

However, the various experiments made in the course of time to discover the etiology of gastric ulcer cannot all be enumerated here. It must suffice to call attention to the fact that in the development of ulcer we are dealing either with the consequences of a mechanical, a chemical, or a thermic lesion of the mucous membrane, with a disturbance of the circulation at the point of lesion, or else such disturbances—and this is more frequently the case—have arisen without a preceding coarse lesion in a circumscribed, vascular area. A limited destruction of the tissue follows because the gastric juice attacks those areas which are no longer normally nourished, just as, to a fuller extent, it consumes the stomach after death, and under favorable conditions it produces softening. There is always, however, a pathologic disproportion between the composition of the gastric juice and the nutrition of the gastric mucous membrane, and the

changed nutrition may be the consequence of an altered composition of the blood as well as of a change in the eireulation. As this has been touched upon in a diseussion which shows conspicuously the present status of our science, we shall not here enter upon a historie retrospeet. Up to the present time the view has been generally held that both of these causes have been deeisive, first, the inereased produetion of acid (hyperchlorhydria) and, secondly, the circumscribed tissue lesion of the mucosa due to such eauses as have been mentioned. But there is no unanimity of opinion as to whether the hyperacid gastric juice which produees tissue necrosis by injury or otherwise is first present and thus causes destruction, or whether, inversely, a primary lesion of the gastrie mucous membrane exerts a secondary reflex action upon the secreting elements of the entire gastric mucous membrane, so that in consequenee there is subsequently an increased production of acid.

In fact the view that this factor alone is operative is no longer tenable, since it has been shown, as we shall later see, that ulcer of the stomach is not invariably combined with increased excretion of hydroehloric acid, but that even a marked decrease may be observed. Therefore the development of the affection must primarily be attributed to the circulatory disturbance always produced, and to the circumseribed tissue necrosis resulting therefrom.

Unquestionably many gastric ulcers are the direct result of vascular lesions and their consequences,—the hemorrhagie infarct,—and the primary cause is either an occlusion of the smallest arterial vessels arising from the submucosa between the tubules of the mucosa, or atheromatous, amyloid, or aneurysmal disease of the walls of the vessels, or even simple stasis and spasmodie contractions of the muscles may lead to rupture of the vessels. In some eases v. Openchowski found hyaline degeneration of the walls of the finest vessels in the hemorrhagieally infiltrated areas of the mucosa, and this he holds responsible for the development of the latter. Lépine and Bret have described a stomach which at the autopsy was completely filled with blood. The patient, a man aged 65, had repeatedly suffered from hemorrhages (due, as was shown, to small ulcerations produced by an endarteritis of the smallest vessels).

But these causes alone do not explain the numerous cases, particularly in youthful persons, in whom disease of the vessels or the other factors mentioned do not exist. In some of these it must be assumed that the ulcers develop from follieular hemorrhages and the hemorrhagic erosions of Rokitansky, which, in miniature, disclose a condition similar to the hemorrhagie infarct, namely, small areas of the mucous membrane suffering from insufficient nutrition. Insignificant local stasis or slight trauma may be the cause of such hemorrhages. They develop *into hemorrhagic erosions*, circular losses of substance from the size of a millet seed to that of a pea, or narrow, striated abrasions in which occasionally

a blackish-brown extravasation of blood appears simultaneously with a loosening of the mucous membrane. Their number varies greatly. Occasionally they are so numerous in the region of the pylorus that the mucous membrane appears to be covered with them. From this erosion the typically chronic ulcer develops. But certainly not from those erosions which, as Langerhans emphasizes, in my opinion most justly, by their irregular distribution upon the mucous membrane, their numbers, and their confluence, differ widely from the typical ulcer with its characteristic appearance and its solitary seat. Langerhans states, "These hemorrhagic erosions usually develop from spasmodic contractions and coincident inflammatory processes; the round gastric ulcer, after a primary affection of an arterial vessel. Only exceptionally do erosions develop into chronic ulcers." But this author, and D. Gerhardt as well, accurately describe typical examples of the transition of such erosions into classical ulcers, and, as Gerhardt has shown, the small ulcers which develop from the swelling and rupture of lymphatic follicles may here be included.

According to the descriptions of Nauwerk, and the published cases of Letulle, Dieulafoy, Stokes and Krafft, the influence of bacteria, that is, of bacterial invasion, in the development of gastric ulcer, appears, on the contrary, to be of mechanical rather than of a true toxic nature, and we can hardly doubt that the lesions were originally bacterial emboli of the smallest vessels, upon the base of which the ulcer later developed, and this was the primary cause of a limited disturbance of nutrition of the mucous membrane. In the upper layers of the necrosed mucous membrane, various microörganisms collect.

A. Schmidt claims to have discovered an equally important factor for the development as well as for the cure of the ulcer in the possibility of contraction of the musele situated below the lesion in the mucous membrane. Griffini and Vassale had already demonstrated that the healing of erosions of the gastric mucous membrane in animals takes place by the complete approximation of the edges of the wound, and thus further digestion by the gastric juice is prevented. Schmidt believes that the absence of such contraction accounts for the production of an ulcer from superficial losses of epithelium, because in experimenting upon a dog he was able to produce deep losses of substance though not chronic progressive ulcers whenever by suitable measures he prevented the muscularis in the area of the lesion from contracting. The conditions are similar in the human being, and the well known fact that gastric ulcer is so frequent in chlorosis and anemia might be attributed to the flaccidity (atony) of the gastric muscles. Admitting the latter to be true—chlorosis, in fact, promotes relaxation also in other organs, the heart, the intestine, the uterus—Schmidt's view has been by no means generally accepted. Years ago, I produced the same deep losses of substance in

dogs, and, by severing the medulla oblongata, circumscribed hemorrhages into the stomach without the contractile power of the muscularis having suffered; besides, numerous cases are observed whose entire behavior excludes from the onset a flaccid condition of the stomach, and in which there was neither chlorosis nor anemia.

The *relation of the nervous system* to the development of gastric ulcer has been repeatedly studied. Lately Talma and his pupil, van Yzeren, have attempted to prove by clinical and experimental research that a spasm of the pylorus due to nervous influence is an etiologic factor. I have remarked elsewhere that this view, for several reasons, is most improbable; above all, because in the overwhelming majority of ulcers there is not the slightest indication of spasm. In my opinion van Yzeren confounds cause and effect, for he bases the results of vagotomia subdiaphragmatica in rabbits upon conclusions arrived at in man. Spasm of the pylorus, wherever present, is not the cause but the consequence of an ulcer. We have long known that conditions induced by hyperacidity, even where they run their course without producing ulcer, may lead to occasional or permanent spasm of the pylorus. Dalla Vedova has succeeded —as well as other authors before him—in producing necrotic, hemorrhagic, and ulcerative changes in the wall of the stomach of the dog by injuring the celiac plexus or its thoracic roots (splanchnic), the characteristic necrobiotic lesion closely resembling the peptic ulcer of man. From the nature and sequence of the injuries caused—hemorrhages of the gastric mucous membrane from the size of a pin's head to that of a lentil, hemorrhagic erosions, true ulcers and typical cicatrization—it is obvious that ecchymosis may be regarded as the beginning of the process from which other conditions arise.

The *development of gastric ulcer from trauma* has lately become especially interesting to us because the laws in regard to accidents frequently place us in a position where we must give an opinion as to the causal relation between trauma and ulcer. Many more or less trustworthy observations are distributed throughout literature which, in their totality, permit no doubt that gastric ulcer may, indeed, be produced directly by a blow, a contusion, a fall, or the like, which affects the gastric region. A comprehensive study of this aspect of the disease will be found in Stern's book, "Ueber traumatische Entstehung innerer Krankheiten," Jena, 1900.

I have repeatedly been called upon for such an opinion, and the following considerations have been my guide. First, we must ascertain whether immediately, or, at least, a short time after the trauma, an undoubted gastric hemorrhage occurred, for this is the only positive sign of a severe injury to the gastric mucous membrane. Directly following this, nausea, anemia, syncope and localized gastric pain may occur; but since, as is well known, such injuries of the mucous membrane heal

rapidly without sequelæ, it is also necessary that, following this hemorrhage, the typical symptoms of ulcer arise, i. e., gastric pain, particularly after meals, and pain upon pressure in the gastric region. The latter symptoms do not always appear immediately after the trauma but, nevertheless, they must show a certain connection with it. In my opinion the diagnosis is best founded in those cases in which, in the course of time, a decided gastric dilatation or a cancer of the stomach, particularly about the pylorus, develops; the former, because it points to a stenosis of the pylorus, which in the given case may be the consequence of a traumatic ulcer at the pylorus; the latter, because modern experience has taught us the frequent connection between ulcer and carcinoma. (Upon this occasion, I shall only remark, parenthetically, that the *direct* relation between trauma and cancer does not appear to me to be sufficiently proven to warrant us in maintaining this association without the connecting link of a positive or, at least, very probable traumatic ulcer of the stomach.)

Unquestionably many different and obscure factors assist in the development of peptic ulcer. Backmann, who investigated the distribution of gastric ulcer in Finland, and from his experience also described the etiology, maintains that "except for a few cases in which trauma very likely played an etiologic rôle, the etiology was very obscure." In passing it may be remarked that Sohlern's hypothesis, repeatedly combated, according to which vegetable food confers immunity from ulcer of the stomach, is not supported by Backmann's conclusions. The population of Finland subsist chiefly on potatoes, bread, porridge, and peas. Meat is eaten only upon Sundays and holidays, nevertheless gastric ulcer is quite common.

No matter in what manner ulcer may develop, in a pathologico-anatomical respect it is not an "ulcer," but a "progressive tissue necrosis," in which the chief characteristic of ulcer is entirely absent, namely, "the proliferation of young cellular elements which persistently invade the tissue more deeply and invariably cause new elements to appear upon the surface." The ulcer does not grow by an active process in the tissue with subsequent destruction, but by a passive process. The tissue only becomes actively involved by cellular infiltration which leads to cicatrization.

PATHOLOGICAL ANATOMY

In regard to the gross anatomy of ulcer and its consequences a few words will suffice. Its funnel-shaped or crater-like structure, corresponding to the ascending vascular tree, is well known; the edges are at first clean cut, later becoming thickened, often permeated by hemorrhages which are due to the peptic erosion of the smallest vessels. Frequently, especially in the acute perforating ulcer, the form is circular, but it may be linear, oval, or terrace-shaped. The base of the ulcer is usually smooth, or it may

show a few uneven points, occasionally it is covered with bloody coagula or a greenish or brownish tough mucus. The size varies greatly, generally from that of a dime to a quarter of a dollar, but it may be very much larger, and sometimes may entirely cover the stomach. Its seat is preferably at the pylorus and on the greater curvature, which are the lowest portions of the stomach when in the erect posture, and those in which the gastric juice collects, so that Nolte has given the following scale of frequency: At the greater curvature 22, at the pylorus 13, upon the anterior wall 3, upon the posterior wall 2, at the cardia 1.

According to Brinton 42 per cent. of the cases are upon the posterior wall, 26.8 per cent. at the lesser curvature, and 15 per cent. at the pylorus. These reports are based on 220 cases. But such statistics are constantly modified by circumstances. In my experience, for example, ulcers of the pylorus are more numerous than those of the lesser curvature, and these are more frequent than those of the greater curvature. Gluczinsky coincides, for in 95 autopsies he found 63 cases of ulcer in the pyloric region. In the majority of cases there is but one ulcer; several, that is, three or more, are rare. Lange, however, in one case saw so many "that he was obliged to discontinue counting them."

Occasionally several originally distinct ulcers coalesce and form one large one.

From *microscopic sections* through the margin of a fresh ulcer the glandular tubules are seen trough-shaped, as if sharply cut off, descending toward the floor of the ulcer.

They are digested in so far as the tissue is unable to resist the peptic power of the gastric juice. In older ulcers a reactive inflammation sets in at the periphery, and leads to the formation of a calloused margin. Here the supporting fibers between the resisting tubules are thickened, and partially formed into oblique layers. The glandular epithelium, in so far as it is still present in the fundus of the glandular nests, has suffered a peculiar change. In place of the lab-cells, cubical or cylindrical epithelia have appeared. These masses are contracted so that they are separated from the membrana propria and from one another. Their nuclei cannot be recognized by staining, their contents have a friable, light, glassy appearance like that of hyaline degeneration. The individual tubules show cystic degeneration. The submucosa is permeated by a profuse small cell infiltration and a strong vascular net. The layers of the muscularis are separated by a fibrillary, intermediate tissue, consisting of meshes torn asunder, some of which have been destroyed. We see, therefore, that the entire necrotic process upon borders and floor is surrounded by a zone of irritation, which in its further development leads to true cicatrization, in that the floor of the ulcer constantly becomes adherent to the layer of tissue beneath, and the mucous membrane of the border of the ulcer retracts into its floor.

In the interstices of the glands, not only in the immediate vicinity of the ulcer, but also in those more distant, the mucosa invariably shows massive small cell infiltration, but nothing peculiar to ulcer, only what is found in all inflammatory irritations of the mucous membrane from a mild catarrh to an acute phlegmonous gastritis. I have rarely examined a gastric mucous membrane, no matter whether the patient suffered from a gastric affection or some other chronic disease, without discovering here and there at the base of the mucosa disseminated foci of small cell infiltration (not lymph-follicles by any means!) which had separated the glandular tubules from each other. In fact, the ulcer subsequently produces an irritation in the surrounding areas of the mucous membrane which to a varying extent and intensity implicates this tissue.

The outcome of the necrotic process is especially interesting. We must differentiate between the following conditions:

1. **Cicatrization.**—Here there is a marked difference from the cicatrization produced artificially in the animal. For while these cicatrices, as Cohnheim reports, and as I also found, healed by the restitution of the normal mucous membrane tissue, in man a fibrous cicatricial tissue with a central depression and the well known tendency to contract develops. Thus, striated cicatrices form and produce a distortion of the gastric wall, particularly when the cicatrix is fixed in position by a preceding adhesion with neighboring organs. Girdle-like constrictions form which give the organ an hour-glass or a cucumber shape. Peculiar cicatricial bands or bridges develop, and form a many-celled sac, of which Cruveilhier gives an excellent illustration in his Atlas.

2. **Progressive Corrosive Necrosis.**—This process, provided cicatrization does not occur, continues as long as peptic gastric juice is formed, and finally terminates spontaneously, i. e., by the complications which arise. These are:

(a) *Erosion of Vessels.*—According to the seat of the ulcer and its extent and depth, sometimes smaller, sometimes larger, lumina of the vessels are eroded. The slight tendency to permanent thrombus formation is characteristic, and is probably due to the corrosive effect of the gastric secretion. Among the larger vessels the pancreatic artery, the gastric artery, and the renal arteries, or the corresponding veins, are attacked.

Severe hemorrhages follow which are only terminated by thrombotic coagulation of the blood. If the thrombus is situated in the splenic vein it may produce acute enlargement of the spleen.

(b) *Adhesions to Neighboring Organs and Perforation.*—If the necrosis extends to the serosa, either a reactive inflammation and adhesion to neighboring organs with consequent implication of these, a perigastritis, or, if this be impossible, direct perforation into the abdominal cavity, is produced. Through the corresponding intermediate tissues secondary perforations may take place into the pleural and cardiac cavities. All of the

neighboring organs, the liver, the gall-bladder, the panereas, the spleen, the diaphragm, the heart, the lungs, and the intestines, according to the seat of the ulcer, are exposed to this danger. Occasionally there are adhesions to all of the neighboring organs in the abdominal cavity without an actual perforation, such a condition having been described by Budd.

Finally the tubercular and syphilitic ulcers are to be mentioned.

Tubercular ulcers, which, so far, have always been found in combination with tubercular lesions of others organs, are characterized by their thickened, infiltrated, wall-like edges. Their floor is usually granular, and of a light yellow color. They are pale and, as in the cases of Eppinger, rise sharply from the darkly stained surrounding areas.

In the margin and upon the floor we find tubercular nodules with characteristic giant cells. The ulcer may be solitary or multiple, and only affect the mucous membrane and mueosa, or also the muscularis. Microscopic examination shows the well known tuberculous foci with numerous tubercle bacilli. In some cases (Litten) the serosa forming the floor of the ulcer was permeated by miliary nodules. In Litten's case the ulcer was quite large, 4.2 by 3.3 ccm., its border sharp and serrated, partly swollen and hemorrhagically infiltrated, the remaining digestive tract free from tuberculous ulcerations, but these were present in the larynx, bronchi and lungs. Similar cases have been reported by Talamon-Balzer, by Giles Saburin, and two by Eppinger. An excellent description of a tubercular ulcer is given by J. H. Musser. The case was that of a negro, 44 years of age, suffering from pulmonary tuberculosis and ill-defined dyspeptic symptoms. At the autopsy a gastric ulcer was found, $1\frac{1}{2}$ by $3\frac{1}{2}$ inches, and undoubtedly of tuberculous nature. Many miliary tubercles and much caseous material were found upon its floor, and tubercles in the neighboring submucosa. In this caseous mass were tubercle bacilli. The case of Bar-Bacci is similar: Isolated, tuberculous ulcerations of the stomach with primary, pharyngeal tuberculosis.

Musser mentions a few similar cases, mostly in children, in which also tubercle bacilli were found in the ulcers. Evidently these findings are rare, for in 567 cases of tuberculosis of the intestine Eisenhardt found gastric tuberculosis in only one case, and in nearly 2,000 autopsies of tubercular individuals Simmonds found only eight cases. Marfan, in a study of gastric disturbances in pulmonary phthisis, mentions but a few examples, and these are somewhat doubtful.

In children the affection appears to be more frequent, for in 297 tubercular children O. Müller and Hecker found 8 cases of tuberculosis of the stomach, and Biedert collected 41 cases of which one-third occurred in children. These ulcerations do not all belong to the type of the corrosive ulcer; on the contrary, some are due to true tuberculous foei or softening with a easeous decomposition of the tubercle, or, at all events, to their combination with the corrosive action of the gastric juice upon the ne-

erosed tissue elements. Their point of preference is the pyloric region, and their development may certainly in most cases be attributed to the deglutition of sputum containing bacilli. Occasionally they may have formed upon a hematogenous and lymphogenous base. Such ulcers may also perforate into the abdominal cavity and produce the symptoms of perforative peritonitis. Kundrat, Paulicki, and Struppler have reported typical cases of this kind.

Syphilitic ulcer has no special anatomical characteristics. In the majority of cases that have been accurately observed no opinion is expressed as to whether we are dealing with a primary lesion or with a decomposed gumma. The syphilitic nature of the ulceration has been recognized (Luxenburg and Zawadzki) only by the presence of an endarteritis of the gastric vessels. Engel believes that 10 per cent., and Lang that even 20 per cent., of all ulcers are of a syphilitic nature, but this seems a gross exaggeration. The syphilitic nature of the ulcer is favored by the fact that typical symptoms occur simultaneously with the secondary symptoms of syphilis, and are cured by antisyphilitic treatment (Cullevrier, Schwimmer, Leudet, Murchison, Hayem, Gaillard, Cesaris Demel).

The fact that a *carcinomatous neoplasm* may develop upon the floor of an ulcer, preferably one situated near the pylorus, requires especial mention. This was emphasized by the Vienna School, and proven beyond doubt by Hauser. In my opinion this fact is not affected by the unsuccessful attempt of P. Hirschfeld to prove by statistics that cancer of the stomach occurs independently of the presence or absence of gastric ulcer. Everything can be proven by statistics, but these do not militate against the force of direct observation, and the latter will convince all who have had considerable practical experience (instead of having seen but a few cases) that, in many instances of gastric ulcer, particularly those seated at the pylorus, there is a gradual but uninterrupted transition of the ulcer into cancer. Borrmann, in his excellent monograph upon gastric cancer, in which he differs from Hauser, does not deny this change, as Hirschfeld erroneously assumed. In their ideas of the propagation of the neoplasm into the tissue, these investigators are wide asunder. Clinically, however, these are the cases in which hydrochloric acid production and the peptic function of the stomach continue for a long time, although they gradually decrease toward the norm—cases which, upon the basis of chemism, have been diagnosticated as benign tumor of the pylorus, while operation or autopsy shows a more or less carcinomatously degenerated old ulcer.

It is always difficult to quote statistics in regard to the *frequency* with which definite diseases occur and their peculiarities; rather, to utilize the so-called statistics for nosological and diagnostic purposes. We deal with different classes of patients and different conditions, which to a great extent depend upon unavoidable circumstances. If, however, in the concrete case we attempt to base our opinion upon statistics, we may not know

whether the case is typical or exceptional. It is true that certain circumstances, that is experiences, are so positive and invariable that they require no statistical proof. Among these is the well known fact that women suffer more frequently from gastric ulcer than men, but the reports of authors differ widely as to the exact proportion. Joslin gives the ratio as 1 : 5, Rheinwald as 1 : 1.2. Both authors base their figures upon comparatively few cases, respectively 190 and 187, while in 3,114 cases Murrell found a proportion of 1 : 2.1 and 1 : 3.4. All authors agree that the age in which most are affected is the third decade, i. e., the time between the twentieth and thirtieth years of life. Among 2,164 cases Murrell found 393 between the twentieth and thirtieth, and 265 between the thirtieth and fortieth, years of life. Joslin found the average age in men as 37, in women 27. In Rheinwald's cases 9 males and 31 females between sixteen and twenty years of age, and 19 males and 22 females between twenty-one and twenty-five years of age, were attacked.

It is well known that the proportion in cancer of the stomach is just the reverse, i. e., that more men than women are attacked, and that in later life those between the fiftieth and sixtieth years are most liable; therefore, the comparative youth of the patient in a doubtful diagnosis of ulcer would be of some weight if we had not recently learned by surprisingly numerous experiences that youth is not exempt from cancer. The youngest case of cancer of the stomach that I have observed occurred at sixteen, the oldest at sixty-five years of age. It is true that in the former case carcinoma had developed from an ulcer.

SYMPTOMS

The symptoms of gastric ulcer which authors have described as belonging to the so-called normal type need not be dwelt upon; only the most prominent will be referred to.

Our discussion will be chiefly devoted to the manifold deviations which often make the so-called classical clinical picture appear quite differently from that described in the text-books.

The symptom-complex of ulcus ventriculi may be divided into the following groups:

1. Cases in which the irritative symptoms which result from hemorrhagic erosion or the corrosion of larger or smaller areas of the mucous membrane predominate, and there are no complications.

2. Cases in which the previously mentioned irritative symptoms and hemorrhage simultaneously appear.

3. Cases with symptoms of irritation and perforation.

4. Cases which run a latent course up to the time of death from hemorrhage or perforation.

5. Cases which present the sequelæ of ulcer.

When we remember merely the fact that in these groups the symptoms may be variously combined, an ever-changing clinical picture is presented.

As is well known, we usually diagnosticate gastric ulcer when the classical triad of symptoms is present: Hematemesis which can be definitely traced to the stomach; typical, and usually sharply localized, pain occurring at regular intervals after the ingestion of food; and the increase of free hydrochloric acid in the gastric contents.

From this it is evident that gastric ulcer can only be diagnosticated with certainty in advanced stages, and can only then be recognized if the previously mentioned symptoms occur in combination. *This, however, in my experience is by no means invariably the case, and I might even say that such cases are exceptional.*

As the nerves exposed in the ulcerated surface are irritated by the ingesta and thus the pain is produced, and the muscular contractions which arise in consequence of the irritation may also cause painful sensations, *gastralgia* soon forms an important feature of the clinical picture.

Early in the affection the pain becomes localized in a definite area, usually the seat of the ulcer, and mostly in the pit of the stomach. The boring, sharply defined pain which frequently extends antero-posteriorly is characteristic. It usually increases upon pressure, and its intensity may be determined by the so-called algesimeter which is employed by nerve specialists, without the diagnosis becoming any clearer or any points of support being gained as to the localization (Boas). Women cannot lace, and men cannot wear a firm belt; only exceptionally is the pain ameliorated by pressure. It is obvious that the pain is most intense when the raw, ulcerated surface is exposed to great mechanical irritation. First, and most frequently, that after eating, either because the ingested food directly irritates or by its weight distends the gastric wall, that is, causes contraction and spasm, especially if the ulcer is located at the pylorus. This is relieved as soon as the stomach rids itself of its contents; as, however, small or coarse particles of food are occasionally caught in the grooves of the ulcer and may remain there, it is not remarkable that the organ to a certain extent reacts to these irritative bodies, and repeatedly makes an effort to expel them. Thus spasmodic contractions, *pylorospasms,* which may last for days or may recur at short intervals, arise and greatly weaken the patient by their duration and the suffering they cause.

These are generated in highly developed processes of stenosis of the pylorus or of great atony of the muscles of the stomach by the retention of acid products of secretion, with the starches which are feebly or not at all digested. Distention of the stomach from gas and irritation of the nerves by the continuous ulcerative process produce gastralgia, which must be looked upon rather as a reflex effect when cold and psychical stimulation produce gastralgic attacks, or the pains increase in severity before the menstrual period, and lessen when it appears.

These, however, are not the only causes. In patients with peptic ulcer I have repeatedly seen severe gastralgia after a cold drink or a spoonful of hot soup, a swallow of too hot tea or any similar thing; in these instances the previously mentioned factors were out of the question, but a thermic irritation was the cause. In my experience too hot ingesta is a cause much more rarely than cold, perhaps for the reason that with the former, the mouth and pharynx to a certain extent act as gourds, the gastric mucous membrane is more tolerant to high temperatures than to low ones, and also because less is consumed of the former than of the latter.

Gastralgia accounts for the fact that these spasmodic pains usually come on suddenly and with great intensity, and that they disappear almost as rapidly as they occur, and the patient quickly returns to nearly a normal condition, while a gradual increase to a paroxysmal height is rarely heard of.

The occasional occurrence of cutaneous hyperesthesia and anesthesia, as was observed by Traube, is peculiar, and is attributable to central irradiation. Attacks of angina pectoris, of intercostal neuralgia, of neuralgia of the left brachial plexus, of sensations of pain in the right shoulder (Brinton) and in the arms and legs (Müller) appear. Pressure points are noted upon the back, and along the vertebral column, corresponding to the posterior roots of the lower thoracic and upper lumbar intercostal nerves, or a spontaneous pain, or one after pressure, appears in the spinous processes.

In opposition to some recent investigators, particularly Pariser, I maintain that these pain and pressure points upon the posterior walls of the thorax *are by no means invariable.* On the contrary, in my patients they were often absent, and, like the author mentioned above, I should be unwilling to base a diagnosis, or the permanency of the cure of an ulcer, upon their presence or absence. As the demonstration of this phenomenon is very simple, errors can hardly result from the method.

At this point *tetany* must be mentioned, which, however, is but slightly connected with gastric ulcer, being not an immediate but only a remote sequel. This peculiar symptom-complex is often attributed to a deficiency of water in the organism, and is characterized by the hyperirritability of certain peripheral nerves, above all, the facial nerve and those which innervate the extensors of the extremities; among other conditions it occurs in gastrectasis following ulcer of the pylorus, and gives rise to stenosis.

Gastric dilatation which, in its course, leads to stasis of the gastric contents and a deficiency of water in the organism, may, however, be due to various causes. Hence, tetany does not clinically belong to the picture of gastric ulcer, but is only a complication which occurs in a slight number of cases. At this point I cannot refrain from expressing my belief that deficiency of water in the organism, brought forth so prominently by Kussmaul's adherents, is the causative factor in only a small number of

cases. Other authors, as well as I, have observed cases of tetany in which there were no signs of gastrectasis and its consequences, but the cause was to be sought for in the increased reflex irritability due to abnormal decomposition of the ingesta and the formation of toxins thereby.

In nearly three-fourths of all cases of gastric disease, there is an "*epigastric pressure point*," i. e., an area in the scrobiculus cordis sensitive to pressure, and this is also present in most patients suffering from ulcer. That this has nothing in common with the localization of the ulcer is a well known fact and experience. To be distinctly differentiated from it is a point lower down and to the left which upon pressure also very frequently, but not so often as the first mentioned one, is sensitive to pressure, and in its position corresponds to the solar plexus. In fact in most persons, with and without ulcer, a decided sensitiveness to pressure may be observed in this area, provided the pressure is exerted obliquely toward the median line, and thereby the solar plexus is forced toward the bony vertebral column. That in thin persons with rigid abdominal walls a distinct epigastric pulsation is felt in this area hardly requires mention.

Only rarely is the accompanying *gastric catarrh* so prominent that actual anorexia, a pappy taste, eructations, a disagreeable odor to the breath, and a *coated tongue* are observed. On the contrary, the latter organ is usually of fair appearance, smooth, moist, and distinctly red, so that the findings just mentioned in patients with severe, colieky, gastric disturbance should at once awaken our suspicions of ulcer. What of the *chemical processes* going on in the stomach?

Here we are forced to touch upon the question so often discussed whether it is permissible, with a certain or suspected ulcer, to introduce the sound or the stomach-tube for diagnostic or therapeutic purposes. Absolutely yes! In many hundreds of patients with ulcer I have introduced the stomach-tube, or have had it introduced, thousands of times, and have *never* seen deleterious effects.

This is maintained by all authors of great experience. Of course, this manipulation is not to be undertaken *immediately* after a profuse hemorrhage. But we shall see later on that during decided bleeding it is very beneficial to wash out the stomach with ice-water.

That in a majority of the cases the chemism is not decreased is evident from the fair appetite of patients with ulcer. Yet the early view that an increased production of hydrochloric acid is a necessary or, at least, an almost invariable accompaniment of gastric ulcer can no longer be maintained, being contrary to the comprehensive observations of the last few years. In my cases I found hyperacidity in only 34.1 per cent., normal acidity in 56.8 per cent. and subacidity in 9 per cent.; I must admit that these results surprised me greatly. It must be remarked, however, that in the last mentioned group there were five cases in which the acidity on repeated investigations was found to be either 14 per cent. or 24 per cent.,

while the presence of a typical gastric ulcer without carcinomatous degeneration was made certain by an operation and the subsequent microscopic investigation of a resected portion of the tissue. It is true that in the other cases of this category we were dealing with persons in whom a more or less advanced degeneration was either assumed or was determined positively by operation.

Rheinwald tested the chemistry of the stomach in 66 cases, and found hydrochloric acid in 84.5 per cent., hyperchlorhydria in 65.5 per cent., an absence of hydrochloric acid in 7 per cent., lactic acid 5 times. The frequency of non-increased, and even subacid, values has also been noted by other authors.

In 38 cases of ulcer Schneider found hyperacidity in only 18 to 19 per cent. The reason he assigns for this is that simultaneously complications of various kinds arose, such as ectatic conditions, chronic peritonitis, more or less severe anemia, etc.

Köhler, who observed the cases in Gerhardt's Clinic from 1890 to 1895, found among 165 tests for hydrochloric acid 65 per cent. of positive, and 34.7 per cent. of negative results. It is true the extreme proportion of lactic acid, namely, 33.3 per cent., forces us to conclude that in many of his cases there was already well advanced carcinomatous degeneration.

According to these experiences, the opinion that all gastric ulcers during the time of their existence cause a typical increase of acidity, must at all events be rejected as incorrect.

On the contrary, here—as in many other points in the diagnosis of gastric disease—we must be very cautious in utilizing the chemical findings. Increased acidity favors ulcer, its decrease does not positively exclude it.

In fact, upon repeated examinations the values for acidity show decided variations. In 35 cases, in which I was not content with a single test but made repeated examinations, sometimes six or seven, the values in some respects differed widely from each other. Thus, in a woman, aged 25, who several times suffered from hematemesis, and in whom the gastric contents after a test breakfast frequently showed small quantities of blood, the values for acidity varied between 28 and 44; in a patient aged 26 between 36 and 78; in a man with repeated hematemesis, and who perished from perforation, between 38 and 54; in another patient, in whom resection was performed for ulcer at the pylorus, between 29 and 71, etc. Treatment, and particularly the diet, influences these variations, but they are especially important because these are usually the cases in which we resort to the stomach-tube to obtain a positive finding, if possible. Köhler also states that in some cases of undoubted ulcer of the stomach a number of tests are apt to be negative until free hydrochloric acid is produced in large quantity, and sometimes no hydrochloric acid can be found with

any tests. *Riegel's statement that hyperacidity is a constant sign in ulcer of the stomach must, therefore, be accepted with limitations.*

Gluczinsky has called attention to the fact that chemism changes when a correct diet is instituted, that is to say, changes with the food. For example, in the first week on a mixed diet an acidity value of 30 (hydrochloric acid) was found after a test breakfast, in the second week upon a milk diet 8, in the third week upon mixed diet 25, and in the fourth week upon a milk diet 5. In the cases which I cited it is unnecessary to consider this circumstance; for, during the entire time the patients were under observation, they were kept upon the same bland diet.

In a certain group of cases, however, an increase in the excretion of hydrochloric acid is unquestionably present, and these are the cases designated as *gastrosuccorrhea* or *hypersecretion* (parasecretion, Ewald). Formerly relegated by many authors to the true neuroses, they are now regarded as the consequences of ulcer running a latent course. Soupault operated upon 28 cases of typical gastrosuccorrhea, and in each instance found an ulcer of the pylorus. In 48 cases of gastrosuccorrhea Mathieu and Laboulais found hematemesis or melena six times, and eleven times very severe pains were referred to spasm of the pylorus. Gluczinsky attributes all cases of gastrosuccorrhea to ulcer with stenosis of the pylorus. This, in my opinion, is to act without discrimination. The outpouring of gastric juice is occasionally irregular and unsteady. It is unreasonable to assume in each case a newly formed ulcer, but in all of these instances we must consider nervous irritation as a cause, as well as the fact that these cases cannot be strictly separated into *"retention secretion"* and *"hypersecretion,"* nor do they belong exclusively to one or the other category. Every one of extensive experience knows that transitions occur, and that the same case may at certain times show the characteristics of a pure neurosis, and at other times may present the picture of deranged stasis. Therefore, explicit descriptions to show that either the one or the other condition was alone decisive and causative appear to me to be quite superfluous. Yet this is now a secondary question. I desire only to show that certain cases of ulcer positively present increased secretion of hydrochloric acid, and, disregarding the limitations mentioned above, the latter, i. e., hyperchlorhydria, is unquestionably of great diagnostic value.

I go a step further, and maintain that in all cases in which the course points with more or less likelihood to gastric ulcer, the chemism of the stomach, however, showing insufficiency of hydrochloric acid secretion and of peptic digestion, the view of a carcinomatous transition of the ulcer is well founded. This is particularly true in young persons. In this respect I have recently seen a most typical case: A woman, aged 26, had a large, freely movable tumor situated upon the greater curvature and toward the pylorus, the mucous gastric contents showing undigested remains of a roll but without free hydrochloric acid. Operation revealed two flat

ulcers the size of a twenty-five cent piece, one close to the pylorus, the other toward the fundus, in the surroundings of which the submucosa, the muscularis and serosa were markedly thickened. The floor of the ulcers was almost smooth, of a light flesh color, the margin was slightly raised, but not undermining the neighboring, greatly swollen mucous membrane. Microscopical investigation showed a carcinomatous neoplasm extending into the muscularis, which only in quite isolated areas had left some glandular tissue. In this case the history revealed no former symptoms of ulcer, but the age of the patient was against a primary carcinoma. In other similar cases, however, typical signs of an old ulcer could be gleaned from the history.

The hemorrhages are either from small vessels and slight in amount, and then are only accidentally observed in the form of fine hemorrhagic streaks when freshly admixed with the vomited material, or they are seen as reddish-brown, granular masses after the gastric juice has for some time acted upon the accumulated blood. Upon superficial examination small hemorrhages of this kind may be entirely overlooked, for the blood is not vomited at all, but passes into the intestine, and is there so decomposed that the appearance of the feces is not decidedly altered. Slight admixture of blood to the gastric contents does not give it a characteristic appearance, but often only a dirty gray color, which does not at once awaken the suspicion of hemorrhage. On the other hand, it need hardly be stated that cocoa, chocolate, strawberry wine, bilberry wine, and the like may lead to errors upon superficial examination. In such cases, as Schmauss has shown, we must occasionally make microscopic, spectroscopic, or chemical examinations in order to detect the presence of the smallest quantities of hemoglobin or of blood in the gastric contents or in the feces, and to recognize them as the cause of so-called essential anemia.

Occult Hemorrhage.—In latent gastric ulcer Rossel, and subsequently Boas and I, repeatedly made the diagnosis of ulcer from the proof of "*occult*" blood in the feces, naturally, when other sources of hemorrhage were excluded, and the food administered prior to the examination did not contain large amounts of blood.

The *chemical examination* of the feces for blood is best made either with the resin of guaiac or with aloin. In either case a quantity of feces about the size of a hazelnut is rubbed up with water (or a corresponding quantity of fluid feces is taken), and, after the addition of some glacial acetic acid, is shaken up in a test-tube with ether (the previous removal of fat from the feces by shaking with ether is proper, but is by no means always necessary). The hemoglobin which may be present is changed into hematin (methemoglobin) by the acetic acid, and taken up by the ether. To the clear, over-lying ether which is poured off resin of guaiac which has been dissolved in alcohol is added (or the dry powder which is readily soluble in ether), and finally about a cubic centimeter of the

resin of turpentine or Hühnerfeld's reagent.[1] When larger quantities of blood are present, a blue color at once appears; when smaller quantities, this change takes place after a few minutes. The test is made in the same way with aloin, of which a freshly prepared alcoholic solution should be used. In a few minutes the ethereal mixture changes to a beautiful, cherry-red color, and the hemoglobin forms a uniform layer of precipitate at the bottom of the test-tube. According to Boas, the test may be made in the same way as for the reaction of indican; the addition of a few drops of chloroform will make the reaction more distinct, but in my opinion this is superfluous. No matter how the reaction for blood is produced, in those cases which present no other characteristic sign of gastric ulcer the difficulty will be to prove that the blood found is actually from the stomach and not from other portions of the digestive tract. It is self-evident that no blood or hemoglobin should be given with the food.

The *microscopic proof* of small quantities of blood in the feces is always very difficult, because the blood-corpuscles in their migration through the intestine are usually so distorted that they lose their characteristics. In profuse hemorrhages there is no danger of this, but in lesser ones, especially when mercurial preparations or those containing sulphur have been administered, it must be considered.

Copious hemorrhages presuppose the erosion of a large vessel, and they act upon the stomach like an emetic, so that this organ rids itself of its contents. Many patients have distinct premonitory symptoms, flashes of heat, epigastric pulsation, a sense of fulness in the gastric region, great, and apparently groundless, internal disquietude which to a great degree produces depression and anxiety. In other patients the only preceding symptoms are an unusual feeling of illness and complete loss of appetite. The time which the blood remains in the stomach varies greatly, and this also changes the appearance of the vomited material. Sometimes the coagulated blood is light red and lumpy, at other times it forms dark red masses, and sometimes, in a minority of cases, the vomited material resembles coffee-grounds. Large quantities of blood in the vomited material may even be noted with the naked eye, certainly by the aid of the microscope, perhaps by the spectroscope, or by the various chemical tests for blood. As mentioned above, a portion of the blood finds its way into the intestines. If a profuse hemorrhage has occurred, or if the ulcer is in the duodenum, the evacuations have a tarry appearance, and consist of very offensive masses in which the presence of blood may be detected.

In most cases the hemorrhages occur suddenly without an assignable cause. Occasionally they are preceded by circumstances either of a psychic or a physical nature which accelerate the cardiac action, such as joy, fright, unusual exertion, external trauma (blow, fall, pressure, etc.), or

[1] Acid. acetic., Aq. destill. āā. 2.0, Ol. Terebinth., Spirit. dilut. āā. 100.0.

strain upon evacuation. Improper food, for example, hard tendinous meat, fibrous vegetables, such as beans or asparagus, may cause hemorrhage by direct mechanical lesion. One of my patients, who up to that time had never vomited blood, had a severe hemorrhage after taking, on the advice of Cohnheim, 200 grams of oil by the mouth to relieve pyloric spasm. Nausea, retching, and hematemesis were the consequences of this form of medication which is greatly praised by its author.

The frequency of hematemesis is reckoned at 50 per cent., but this is too high rather than too low. Brinton gave 29 per cent., Witte in Copenhagen found it 100 times in 339 cases (= 29.4 per cent.), Gerhardt saw it in 47 per cent. of his cases, so that we may safely assume that more than half of the patients do not suffer from hematemesis. Among 556 cases v. Leube found it in 46 per cent. I observed it in 54.5 per cent. In 187 cases Joslin found no less than 81 per cent. showing gastric hemorrhages, from which we may see how unreliable and how dependent upon accidental conditions these so-called statistics are.

In an interesting compilation based upon the records of Guy's Hospital from 1870 to 1890, W. Charles Hood reports that in the majority of cases of gastric hemorrhage in the course of ulcer occurring in patients under 30 years of age women are chiefly affected, and that during this period of life fatal hemorrhage is extremely rare. Of 66 cases of this kind 29 were under thirty, among these only 2 men. On the other hand there were 11 men among 21 cases between thirty and forty years of age. All recovered. Seven other cases in which death occurred immediately after the hemorrhage were all over 30 years of age, among them 4 women, respectively 33, 35, 50 and 53 years of age. Although we can hardly assume that the prognosis of gastric hemorrhage changes particularly with the critical age of thirty in women, nevertheless this report indicates that gastric hemorrhages in younger women present a less unfavorable prognosis, since a conspicuous improvement, the cessation of the distressing symptoms and regeneration of the blood, is often observed after such an event. In one of my cases the number of erythrocytes within three weeks after the hemorrhage increased from 1,900,000 to 3,040,000, and the amount of hemoglobin from 31 per cent. to 51 per cent. Similar observations have been repeatedly made.

The hemorrhagic masses from a bleeding peptic ulcer are free from specific elements, and the blood-corpuscles preponderate to such an extent that the cellular elements of the gastric mucous membrane are either scant or do not appear at all.

When a copious hemorrhage has once occurred the danger of a repetition is always present and hangs, like the sword of Damocles, over the patient's head in a two-fold manner. In the first place, at brief intervals in the course of a day, or even several times a day, or, perhaps, at brief intervals during a week, hemorrhages repeatedly occur. We must then

assume that we are dealing with relapses from the same vessel which was first eroded. Secondly, after longer intervals, months or even years, hematemesis may be renewed, and then, in consequence of the tendency of the individual to hemorrhages of this kind, repetitions are likely. Occasionally it appears as though the thrombus formed is not adherent, and that it readily sloughs off when the cardiac action becomes stronger, as under normal conditions. Twice I saw a hemorrhage occur after a long interval when the patient, from a mistaken idea that it would be beneficial, took strong alcoholic liquors, although only in small quantity.

Slight hemorrhages, aside from their psychical effect, have no influence upon the condition of the patient; copious hemorrhages, particularly when in rapid succession, lead to extreme anemia and its consequences. Wax-like pallor of the skin, a small frequent pulse, slight fever, tinnitus aurium and vertigo, loss of consciousness, transitory mild delirium, and total anorexia follow. Subsultus tendinum and spasms in the extremity like those of cholera have been observed. Nevertheless, the patients recover with comparative rapidity, and under suitable treatment the lost strength is regained. Edema of the extremities, which is observed particularly in the evening if the patient has been upon his feet during the day, and amaurosis which sometimes occurs immediately, perhaps some time after the hemorrhage (but which, according to Fries, occurs in 65.5 per cent. of all hemorrhages in the intestinal tract), show an internal relation to hematemesis which as yet has not been clearly demonstrated. As has already been stated, cases of gastric hemorrhage with fatal outcome, particularly in youth, are comparatively rare.

Here also the reports of authors differ widely, and the results vary between 8 per cent. (Rodman) to 0.8 per cent. (Bramwell). My own statistics, based upon 360 cases observed in the hospital, give 1.2 per cent.

Usually death is due to the rupture of the ulcer and corrosion of the splenic or pancreatic artery, the portal vein, or the left heart, as will soon be described. A case of chronic gastric ulcer with fatal hemorrhage from an eroded left renal vein has lately been reported by A. Markel.

A small aneurysm of the coronary artery was the cause of death in a case described by Powell. The ulcer was situated in the lesser curvature close to the cardia; in its center was an aneurysm about the size of a pea which had ruptured, and by profuse hemorrhage had caused death in a few moments. A similar case was described by Sachs. Here a small artery of the submucosa was implicated.

The hemorrhages which take place by the perforation of an aortic aneurysm into the stomach or into the esophagus are of a more indirect nature. Gastric hemorrhages have also been observed (Naunyn) in cholelithiasis. Minkowski reported a remarkable case at the Congress of Internal Medicine in 1902. It was that of a small aneurysm at the arch of the aorta which had ruptured through the walls of the esophagus.

Thence the blood found its way to the cardia and to the floor of an ulcer in the stomach.

Any of these hemorrhages may occur without preceding symptoms of an ulcer of the stomach. Fatal hemorrhages are usually preceded by indistinct signs of a severe disease; in other cases, however, the hemorrhage may occur suddenly like lightning from a clear sky, attacking a person apparently in the best of health, and bringing about a fatal termination.

At this point we must call attention to other forms of "gastric hemorrhages," which, because less familiar, may tend to grave diagnostic errors.

First, are the *hemorrhages from varicose veins of the esophagus.* Here the condition is such that occlusion of the hepatic circulation takes place, and the blood in the portal vein attempts to force its way through the venous plexus formed by the combination of the gastric veins with the esophageal veins. The blood then takes a retrogressive course, and, as the esophageal veins cannot withstand the force of such large amounts of blood, they become enormously dilated. The least resistant areas dilate and form varices, which, with the great pressure of the blood, readily rupture and cause profuse hemorrhage. The blood at first flows into the stomach and is vomited as a "gastric hemorrhage." Only a thorough investigation and the consideration of the just mentioned possibility will furnish a clue to the true situation. Frequently this is revealed only at the autopsy. I have seen a great number of cases of this kind in which grave errors were made. Only recently a gentleman from the provinces was sent to me with the diagnosis "hematemesis from an ulcer of the stomach"; examination revealed a decided enlargement of the spleen and a hard, enlarged liver. In the gastric contents there was a marked decrease of free HCl; no enlarged glands. The patient had twice suffered from a decided gastric hemorrhage which had appeared without prodromes. Typical symptoms of ulcer were never present. Under these circumstances a diagnosis was made of hepatic cirrhosis with hemorrhage from the esophagus, and this was soon confirmed by the development of marked ascites.

In this group we must include the hemorrhages *from varices of the gastric mucous membrane* (Sachs, Letulle, A. Cohn), which often recur in close succession, and "are combined with venous stasis phenomena of the abdominal organs, often with hepatic cirrhosis, and almost invariably with decided enlargement of the spleen."

In regard to the splenic tumor in these cases, it must be borne in mind that, under some circumstances, as has been previously stated, thrombosis of the splenic vein from a corroding ulcer of the stomach may cause acute enlargement of the spleen. If the thrombus be simultaneously affected, and if, in consequence, a septic fever develops, the diagnostic perplexity is less great.

Another form of gastric hemorrhage not originating from ulcer is the

so-called *"parenchymatous gastric hemorrhage."* There is also a group of hemorrhages which are not limited to the stomach, but attack the entire intestinal tract, and occur as the consequence of venous stasis in diseases of the heart, the liver, and the lungs, particularly in thrombosis of the portal veins, and in infectious diseases, such as enteric fever, yellow fever, cholera, etc. As a rule, they do not lead to typical hematemesis, and scarcely ever simulate gastric ulcer. The condition is quite different when the bleeding appears suddenly, as a single or repeated attack of hemorrhagic vomiting, and the most minute investigation fails to reveal the source of the hemorrhage. As many cases of gastric ulcer run a latent course prior to the appearance of severe hematemesis or melena, we will not err in such cases, after excluding the previously mentioned causes of hemorrhage—here, in my experience, hepatic cirrhosis takes precedence—if we assume a gastric ulcer, and in the further course this view is confirmed by the fact that, soon or late, the classical picture of gastric ulcer develops. Now and then, however, the course is exactly the opposite of this. The hemorrhage soon ceases; for a few days or weeks, although great debility remains, the patient feels perfectly well, and shows no gastric symptoms. On the other hand, repeated hemorrhages may lead to collapse and death. At the autopsy, except for the occasional but by no means invariable superficial erosions of the mucosa, no pathological changes are found which are either directly or indirectly related to the hemorrhages. There are quite a number of such cases in literature—I have seen five such, some of which were published by Reichard—in which the most careful microscopic examination did not show the origin of the bleeding. In a case of Hampeln's, a few erosions of the mucosa were found near the pylorus and the lesser curvature, but, upon injecting the gastric artery, no fluid passed through the eroded areas of the mucosa. In one of my cases the hemorrhage was due to extreme irritation of the gastric mucous membrane, the drinking of a hot infusion of red wine, chamomile, thuja, and various spices. Under some circumstances nothing remains but the assumption of a special permeability of the vessels which are unable to resist the local hyperemia. A case described by Hirschfeld occurred in an old, cachectic woman in whom no change except a very marked arteriosclerosis of the vascular system could be found. Minkowski reports a case of amyloid degeneration of the heart and gastrointestinal vessels without a similar change in the large glandular organs (liver, spleen, kidneys) leading to parenchymatous gastric hemorrhage.

Menstrual gastric hemorrhages, and those occurring *vicariously* in place of menstruation which sometimes may appear intermediately between two periods, usually belong to the group of so-called occult hemorrhages. Actual hematemesis is not observed. On the contrary, the blood is discovered accidentally, or is found while evacuating the gastric contents (Kuttner). Therefore, this condition may very readily be overlooked;

nevertheless, it exists, and v. Schrötter, Sr., only placed his temperamental ignorance on record when he chose to designate my report at the Congress of Internal Medicine (1902) as a canard ("Räubergeschichten")! Whether simple stenosis of the pylorus may be the cause of the hemorrhage, as was assumed by Lambotte and subsequently by Moser (retention of gastric contents, venous hyperemia of the mucous membrane, and muscular contractions) appears to me very doubtful when we contrast this with the numerous cases of pyloric stenosis without hemorrhage. The cases reported by Lambotte certainly do not permit us to exclude the possibility of ulcer. At this point, too, the occurrence of profuse initial hematemesis in gastric carcinoma must be mentioned (E. Mey). In such cases the assumption is obvious that an ulcer originally existed which must have undergone carcinomatous degeneration. In the 4 autopsy reports communicated by Mey, the nodular character of the neoplasm favors a primary carcinoma, for, in the transition of an ulcer into carcinoma an infiltration of the tissue is much more likely to occur. At all events, in such cases the possibility of this must be considered.

The circumstances are different when hemorrhages occur as complications of severe purulent processes or marked circulatory disturbance in the intestinal vessels. Surgeons, for example, v. Eiselsberg, have for a long time called attention to the fact that after abdominal operations, particularly for strangulated hernia, gastric hemorrhages may occur. These are attributed to displaced thrombi from the point of operation. But I explain the condition in the following case differently:

A man, aged 47, was suddenly attacked with severe pain in the umbilical region; there was absolute constipation. After twenty-four hours an operation was performed, at which a diagnosis of internal incarceration was made. Prior to the operation the stomach was washed out, and large quantities of a dark, brownish-red fluid were evacuated; this proved to be almost pure blood. At the operation a portion of the small intestine amounting in length to 1 meter and 30 cm. was found to be constricted by a band reaching from the sigmoid flexure to the root of the mesentery. The constricted intestine was enormously distended, and dark bluish-red in color. The circulation was not reestablished in the constricted portion of the intestine, hence it was resected. The patient lived only twenty hours after the severe operation. The autopsy revealed a greatly distended stomach; the leaflets of the diaphragm were forced up to the fourth rib. "In the stomach countless erosions of the mucous membrane of the size of a pin's head were found. Some of these were round, others angular or elongated; in isolated cases, they extended to the submucosa; at other points they were more shallow. Some contained greyish-red blood, particularly about the borders and at the base; other erosions contained no blood. *A true vascular lesion* was nowhere to be found. The peritoneum near the gastric region showed no abnormalities." (From the autopsy report of Dr. Oestreich.) But little muco-hemorrhagic contents were found in the intestine; the vessels were markedly injected. The abdominal cavity contained about 200 c.c. of a hemorrhagico-serous fluid. In this case we probably find the cause and anatomical foundation for the hemorrhage in the altered circulation of the abdominal cavity, and in the erosions of the mucous membrane which were apparently of an earlier date.

Severe hemorrhages into the stomach and the intestine occurring in the course of septic processes which affect the abdominal organs without an apparent anatomical lesion, and which cannot be attributed to the formation of thrombus, have until recently been observed only in isolated cases. Dieulafoy mentions them in perityphlitis, and regards them as the result of toxin infection of the gastric mucous membrane, the latter, in his cases, showing no ulceration in the region of the pylorus. Guyon has reported a case of sepsis in the urinary passages without any injury to the urinary organs. Such cases may present insurmountable difficulty in the diagnosis, as will be noted from a case which I recently observed.

A man, aged 37, who had been ill for four days, was awakened during the night with pain which soon spread over the entire abdomen. He stated that he had previously suffered from gastric difficulty, and that upon the second day of the disease a chill with rise in temperature to 103.2° F. had taken place; except for this there was no fever during the entire course of the disease. I found the patient with all the signs of diffuse peritonitis,—slight dulness in the right and left inguinal regions. The pains varied, being both spontaneous and upon pressure, and were said to have first appeared in the pit of the stomach. During the night the patient vomited large quantities of a blackish-brown mass containing blood. The vomiting recurred in the course of the day and also during the lavage with ice-water which was later undertaken, the water each time evacuated containing blood. The slight quantity of feces discharged from the large intestine after enemata showed no blood. Consequently, I diagnosticated a perforating gastric ulcer, and rejected the possibility of a perforative perityphlitis on account of the hemorrhagic gastric contents and the original seat of the pain in the scrobiculus cordis. The surgeon called in consultation was of the same opinion, but, on account of the advanced peritonitis, refused to operate. The man succumbed on the third day of my observation, and upon the sixth of the disease. The autopsy revealed general peritonitis starting from a perforating gangrenous appendix. The gastric mucous membrane was entirely intact. But the intestinal mucous membrane in some areas showed a bright red injection, a velvety swelling, and hemorrhagic contents were still present in the intestine itself.

Composition of the Blood.—Most authors—I mention Laache, Leichtenstern, Reinert, Osterspey (Ewald), and also recently Reneki and Dolmatow—have found the *composition of the blood* (and this quite independently of a possible hematemesis) so changed that a decided alteration in the number of red blood-corpuscles, and in the hemoglobin, and, occasionally, an increase of the leukocytes, was present. But these changes are due to secondary conditions, chlorosis, anemia, etc., and are not typical of ulcer. For well known reasons, the composition of the blood immediately after a hemorrhage will invariably be found changed, i. e., the red cells and the hemoglobin are decreased, and the leukocytes slightly increased.

Vomiting.—The next symptom of importance is vomiting.

Vomiting usually occurs after eating. It is caused by an irritation of the walls of the stomach, particularly of the exposed ulcer surface, from the food, that is, the hyperacid gastric contents. This is partly due to

the fact that the strong acid causes a spasmodic contraction and closure of the pylorus, and thus not only pains but an increased and even antiperistaltic action of the stomach is produced. The food is often but little changed, and is vomited admixed with mucus as is the case in the so-called *vomitus matutinus* of alcoholics. Fungi of fermentation and other foreign cellular elements, with the exception of occasional admixtures of blood, are either rare or absent (sarcinæ). At other times the vomitus is a thin fluid, of slightly greenish color, and very acid, which upon standing forms a pappy precipitate consisting of starch granules, cellular detritus, but only a few markedly digested remains of meat. Periods occur in which there is an increase of the vomiting, and the stomach absolutely rejects food.

It is quite apparent even from the external appearance of the vomitus, particularly if it is ejected a considerable time after the ingestion of food, that the gastric contents are decidedly acid. Upon standing two layers form: The upper is a thin fluid, clear, not foaming, and containing a finely granular precipitate consisting of slightly altered remains of starch, plant cells, and the like, in which, however, there are no, or very few, muscle fibers, that is, remains of meat. The test with Congo paper gives a strong reaction for free hydrochloric acid. As a rule spontaneous vomiting is not frequent. It is most prone to occur when the ulcer is situated at the pylorus.

Perigastritis.—When the ulcer has extended to the serosa, and before complete rupture takes place, an inflammation of the external layer of the serosa with the formation of a plastic exudate and perigastritis occasionally occurs. This may be limited to a definite area, or may extend into adjacent parts, or may change to hard, indurated masses of exudate which upon examination resemble tumors. Associated with this are local irritative phenomena, retching, and even mild fever. Distortion of the stomach may take place and, under some circumstances, it may be very difficult or even impossible to recognize the true nature of these conditions. From their character, they are insusceptible to internal treatment, and form a permanent source of perplexity. (See also p. 185 in the description of cicatrization.)

Perforation.—A severe complication of the disease and, perhaps, of the clinical picture is produced by the *rupture of the ulcer and the implication of neighboring organs.* When the peptic process reaches the external layer of the gastric wall and attacks one of the neighboring organs, this is occasionally manifested by a localized sensation of pain in the region of the organ attacked. As a rule, however, it runs its course without any external sign, so that only when we test the disturbance of function of the organ in question can we recognize that it has been implicated in the process. As previously mentioned, there may be hemorrhages from the larger vascular trunks.

The various conditions which arise for consideration may be readily

reviewed if we remember the topography of the organs surrounding the stomach. A most interesting complication is a rupture through the diaphragm and pericardium into the left heart followed by pneumopericarditis, also into the mediastinum with emphysema of the external skin and the accumulation of combustible gas. West describes a preparation in which an ulcer had attacked the portal vein and led to a fatal pylephlebitis. Cases are described as pyopneumothorax subphrenicus in which an encapsulated abscess containing air had formed below the diaphragm. Osler has described a very unusual case in which rupture into the left heart occurred; nevertheless, the patient lived two days, for the perforation closed during every systole and opened during the diastole. Thus the patient gradually bled to death. Rupture into the pleural cavity may be diagnosticated when it leads to pneumothorax and empyema or to a direct communication with the lungs, and, as has happened, particles of food may actually be coughed up. In a case reported by Müller nematodes were found in the pleural cavity.

Rupture into the colon and subsequent lientery are rare. When, in a favorable case, the ulcer ruptures into the abdominal cavity, the preceding adhesive inflammation between the stomach and the neighboring intestinal wall and the omentum forms a cavity which is a sac within a sac, and prevents the propulsion of the stomach contents into the abdominal cavity. After this peritoneal irritative phenomena appear, with circumscribed pain, distention of the upper abdominal region, and fever, perhaps also severe vomiting. If the adhesions are very extensive, there may be complete stoppage of intestinal activity which, with permanent obstruction and increasing marasmus, leads to death, as in a case reported by Budd.

Perforation into the free peritoneal cavity is by far the most frequent complication, and happens either after preceding adhesion and abscess formation or without these. It may appear slowly and gradually or, on the contrary, the exit of the stomach contents may be slow. Adhesive abscesses then form which may later become encapsulated, or may rupture and produce general peritonitis. Usually perforation occurs suddenly without prodromes or, at least, without symptoms which point to such an accident. Without any cause or after a preceding trauma, such as an accidental blow, or pressure against the edge of a table or when leaning over a window-sill, in riding, after they have eaten heartily, or after or during vomiting, the patients suddenly feel severe pains in the abdomen which cause a feeling of collapse and, in a brief time, the picture of *perforative peritonitis* develops: Distention of the abdomen, obliteration of liver dulness, on even the slightest touch excruciating pain which may have a colicky or paroxysmal character, vomiting, singultus, facies hippocratica, small pulse, and with these symptoms the patient succumbs.

Spontaneous pain is usually referred to the gastric region; the ileo-cecal region is mentioned, that is, is sensitive to pressure, only in duodenal

ulcers. According to the exhaustive compilation of Brunner, in about 90 per cent. of all cases of perforation the history discloses the symptoms of a preceding ulcer, which, upon the average, had been first noticed about 3½ years before. These may, however, be absent, and I remember a case in which a young girl, previously always healthy, collapsed at night while dancing, succumbing to a perforating ulcer.

Such perforation may also be caused by spasmodic contractions of the stomach either from vomiting after medication, or from introducing the finger into the pharynx, as is done by many patients to produce eructation and vomiting, or it may occur after introducing the stomach-tube. Faber describes a case of perforation after vomiting induced by the patient. The normal act of defecation is said by Bouillaud to have also been a cause.

The youngest female patient was nine years old, the oldest seventy-one. The youngest male was seventeen, the oldest seventy-two (Brunner).

The seat of perforation in the gastric wall is usually the greater or lesser curvature, very rarely the pylorus or the cardia. The lesion is circular, with smooth, non-elevated borders, and surrounded by a more or less extensive zone of smooth, cicatricial tissue, which is usually friable so that at operation the suture will not hold but tears through, and the healing of the wound is exceedingly difficult.

Cases of such perforation that have recovered without operation are among the greatest rarities. Brunner collected from literature the records of 17, all of whom were treated with opium, ice, and rectal alimentation. Almost invariably the perforation occurred a few hours after eating, while the stomach was empty.

In these cases there is a justifiable doubt as to the correctness of the diagnosis. In a case described by Harland, as the author himself reports, an ulcer with a simultaneously existing gastrectasis simulated perforation. In this category I must also include the case reported by Spicker of the spontaneous cure of perforative peritonitis in ulcer of the stomach, which was probably nothing but an acute gastric dilatation.

It is certain that the symptoms mentioned above may be absent, yet perforation may have occurred. I have seen two cases in which there was at first no marked distention of the abdomen, no shock or collapse, the pulse was regular, the temperature normal. It was obvious, however, that perforation had occurred. In both instances the stomach contained neither food nor gas, the patient for three days previously having refrained almost entirely from food, and, therefore, the rupture of the ulcer was accompanied only by the signs of intense shock—loss of consciousness, Cheyne-Stokes respiration, absence of pulse, cold skin, etc.—while the abdomen was neither greatly distended nor very painful.

The "disappearance" of liver dulness, in particular, is a very uncertain sign, as this may also be produced by the distended transverse colon

that extends high above the liver, or—in very rare cases—by a dilated stomach adherent to the liver.

Musser and Wharton calculate that perforation occurs in about 7 to 18 per cent. of all cases. If this includes the most marked cases only, even this low figure is, in my opinion, much too high. I have not observed more than 1.2 per cent. of perforations. Greenough and Joslin saw perforation in 3.2 per cent. of all cases. Those serious accidents in which several isolated ulcers in different areas of the stomach simultaneously rupture, usually in the anterior and posterior walls (Lovell-Keays and others), are very rare.

Occasionally a gastrocutaneous fistula forms with an opening into the epigastric or left hypochondriac region or between the ribs. This is a very rare occurrence; nevertheless, Murchison collected 12 reports of such cases. Sudden perforation has repeatedly awakened a suspicion of poisoning and led to unjust criminal trials.

The *nature of the cicatrization is of great importance.* It is obvious that cicatricial distortion may lead to the severest disturbance of the gastric functions, one of which, dilatation of the organ with cicatricial pyloric stenosis, has already been described; this produces a limited pathologic picture. In other cases, cicatricial contraction causes torsion of the nerves of the gastric wall or deformity of the organs, or the function of large portions of the muscularis is lost, or adhesions with neighboring organs form and lead to gastralgia or to functional disturbances which appear under the guise of "dyspepsia" of different kinds, the original cause of which is usually difficult to recognize and from which recovery without operation is, as a rule, impossible. In the course of years I have learned to dread this cicatrization even more than the primary ulcer. Not rarely such patients are erroneously considered as "nervous dyspeptics." If the cicatrix is circular and at about the middle of the stomach, the various forms of *hour-glass* stomach or large sac-like dilatations of the same are produced. In washing out the stomach the curious symptom then appears that the stomach apparently cannot be evacuated. After some time the water injected for washing returns clear, suddenly, however, again becoming turbid and admixed with gastric contents, and this phenomenon may be repeated several times. This indicates either the condition just described or an insufficiency of the pylorus, the contents of the duodenum being regurgitated into the stomach.

Hemorrhagic Erosions.—Einhorn has lately devoted much attention clinically to the previously mentioned *"hemorrhagic erosions,"* which have long been known anatomically, and frequently described. They do not present the classical picture of ulcer of the stomach but dyspeptic symptoms, even decided pain, which increases soon after taking food, and continues from one to two hours; emaciation and weakness appear. The chemism is not specially characteristic. Yet almost always in the water

used for lavage—and authors lay particular stress upon this *constancy*—small reddish-white floeculi are present which, upon minute examination, prove to be desquamated portions of the mucous membrane. They show well retained glands with small cell infiltration and an accumulation of red blood-corpuscles between them.

These reports have been several times discussed (Pariser, Hemmeter, Platter, Ewald and others). That no positive diagnostic value can be attached to these mucous membrane particles which appear in the water after lavage, i. e., that they do not reveal histologic changes characteristic of a definite pathologic type, Leuk proved in my wards after thorough and careful investigation. His conclusions have been confirmed by Cohnheim, Lubarsch, Hari and others.

Elsner examined 35 cases in which inflammatory symptoms attributable to the stomach with a decrease or cessation of the gastric juice secretion were determined only 12 times, i. e., in 35 per cent., and found exfoliations of the mucous membrane either transitorily or permanently in the water used for lavage. Only four of these complained of difficulties such as Einhorn described. I have, however, elsewhere expressed my opinion (*Transactions of the Twentieth Congress for Internal Medicine,* Wiesbaden, 1902) that a *well characterized pathologic picture of hemorrhagic erosions does not exist,* and that every author who has attempted to portray this has presented a type differing from that of his predecessors. Not only the differential diagnosis of true ulcer, but also that between neuroses with hypersensitiveness of the gastric mucous membrane and spasmodic conditions of the pylorus, occasions perplexity. A positive diagnosis in such cases cannot be made from the course and the results of treatment; the methods of treatment which, under various authors, have resulted in a cure of "erosions," for example, lavage of the stomach with a one per cent. silver nitrate solution, will also cure an underlying gastric catarrh or the ulcer, which, as some observations invariably prove, may develop from such erosions. Henschen, in his autopsy reports, mentions erosions in 3 cases of tuberculosis, in 2 cases of nephritis (which I can corroborate, Ewald), twice in mental diseases, once in pneumonia and only once in gastritis, and these circumstances raise the question whether the erosions are not rather the consequence of a general affection than the expression of a local disease. In any case, the diagnosis "erosion" is very convenient, and will therefore probably maintain its position.

Fissures of the Mucous Membrane.—Here a change in the mucous membrane which is said to have its seat at the pylorus must be mentioned: Fissures of the mucous membrane which produce pyloric spasm, just as anal fissures give rise to rectal spasm. In his report upon the curative effect of large doses of olive oil in gastric disease (see under Treatment) Cohnheim discusses these fissures extensively without positively demonstrating their existence. He considers a fissure to be present from the

fact of the rapid recovery, i. e., the cessation of pains, and maintains that the patients' symptoms could not be relieved in such a few days if they were due to well developed ulcer. In my opinion this argument does not warrant a positive diagnosis. For he has neither seen these hypothetical fissures himself, nor does he construct from them a characteristic symptom-complex, nor, finally—and this is certainly remarkable—are there any reports or proofs to justify this in the literature cited by him. In the many hundreds of autopsies in which I have examined the stomach with the closest attention, I have never seen such a fissure. Now, I will not deny that an ulcer may occasionally bear a certain resemblance to a fissure —a fissure is really nothing more than an ulcerated lesion of the mucous membrane—although, as the name implies, it must have more of a lacerated appearance than the ulcer which develops evenly—but it remains to be proven that these fissures are so frequent as Cohnheim believes, that among 24 cases (I exclude the 6 cases of undoubted carcinomatous ulcer) he made this diagnosis four times, therefore in 16.6 per cent. of the cases.

Syphilis and Ulcer.—In 1838 Andral propounded the question, Why do not syphilitic manifestations appear upon the mucous membrane of the stomach as well as upon the mucous membrane of the mouth? Since then this question has been much discussed, and more or less positive cases have been reported by Goldstein, Hiller, Virchow, Leudet, Lancereaux, Fauvel, Klebs, and Cornil. The simultaneous occurrence of gumma and ulcer of the stomach has been reported in only two cases. In others (Frerichs, Drozda, Murchison, Chvostek) cicatrices were found in the stomach and simultaneously general syphilis. Among 100 cases of ulcer, Engel found a preceding syphilis in 10 per cent., Lang in 20 per cent. Julien, in his great "Traité des maladies vénériennes," quite properly, is very conservative. In diseases so frequent as the two in question it must always be doubtful whether cause and effect or mere coincidence is before us, particularly as confusion with ulcerating gummata can by no means be always excluded. Only the result of specific treatment is decisive. Several such cases have been reported, for example, by Hiller and Gaillard, but the latter, who has written the most recent monograph upon this subject, admits that we have no certain proof. Specific symptoms are certainly not peculiar to syphilitic ulcers. Nevertheless, with coexisting syphilis and the signs of gastric ulcer it is advisable to institute specific treatment.

Tuberculosis and Ulcer.—Tuberculous ulcerations of the intestinal canal occur frequently, as is well known, but they are not often combined with ulceration of the stomach, perhaps for the reason that the dissolving gastric juice prevents the propagation of bacilli, whether introduced with swallowed sputum or with the blood. Typical symptoms are not peculiar to tuberculous ulcers of the stomach. Sudden death from hematemesis in consequence of eroded vessels has also been observed.

DIAGNOSIS

The diagnosis of chronic ulcer of the stomach is easy, and, when all of the classical symptoms are present, can scarcely occasion perplexity. When this is not the case it is exceedingly difficult or even impossible. Important diagnostic factors have already been indicated; we may, therefore, be brief. Two other diseases of the stomach, gastralgia, or gastrodynia (as the expression of a functional nervous disturbance), and carcinoma resemble the symptom-picture of ulcer when ulcer deviates from its typical course. It seems advisable to tabulate their important points of difference as follows:

Nervous Gastralgia.	Ulcer.	Cancer.
Tongue varies, often pale, and fissured at the borders or upon the surface.	Tongue dry, red, with white streaks in the center, or smooth and moist, or slightly coated.	Tongue pale, furry, in rare cases very red, dry.
Frequent eructation of odorless gas.	Eructations either rare or acid eructations with pyrosis.	Frequent fetid eructations.
Taste unaltered. Dryness of mouth frequent, sometimes salivation.	Taste unaltered.	Pappy, insipid taste.
Appetite irregular, capricious.	Appetite good in the intervals. Thirst.	Appetite decreased or anorexia. Early repugnance to meat.
Varying sensations in the stomach, sometimes heat, sometimes cold.	Burning sensation in the stomach. Circumscribed boring pain, often radiating posteriorly.	Sensation of weight; drawing pains of varying character, perhaps pain in the shoulder.
Spasmodic, burning pain, independent of food, often ameliorated by the latter or by pressure upon the stomach. Pressure points over the intestinal plexuses.	Pains gnawing, rare upon an empty stomach, usually appearing after eating or upon motion and on assuming positions which dilate the stomach; increased upon pressure. Pressure points upon back.	Continuous dull sensations of pain, periodically increasing to paroxysms, often produced by pressure or increased by it.
The chemism of digestion not especially altered.	Digestion of starches frequently slow; that of meat normal, or even accelerated; usually hyperchlorhydria.	Digestion insufficient; usually absence of free HCl; formation of organic products of decomposition.

Nervous Gastralgia.	Ulcer.	Cancer.
Epigastric pulsation.	.....	Epigastric pulsation only with marked emaciation.
Vomiting irregular, sometimes mucus only, sometimes more or less digested gastric contents, rarely admixed with bile.	Vomiting, as a rule, immediately or shortly after eating, and frequently the first symptom of the disease; very rarely without taking food, vomitus hyperacidas.	Severe and frequent vomiting, often periodical, occasionally also before the ingestion of food; mucoid; when acid, due to organic acids; only occurring during the course of other dyspeptic symptoms; vomitus shows but slight digestion; sometimes cancer cells present.
No hematemesis, except as accompaniment of very rare complications.	Vomiting of light red blood or coffee-ground masses; usually repeated in a brief space of time, occasionally very profuse, followed by extreme anemia and collapse. Compensation with comparative rapidity. Blood in the feces. Occult hemorrhages.	Decomposed blood more frequent than fresh; quantity usually slight, but, having once appeared, recurring frequently at short intervals.
Almost invariably stubborn constipation; normal evacuations very rare; occasionally fluid mucoid dejecta, the so-called pseudodiarrhea. Mucous colic, that is, colitis mucosa membranacea.	Bowel discharges vary, not infrequently diarrhea in consequence of intestinal irritation. Lientery after perforation into the colon.	Almost invariably bowels stubbornly constipated. Lientery after perforation of the colon.
No fever.	Mild fever only with adhesive inflammation after rupture of the ulcer, or following profuse hemorrhages.	Fever rare, and only toward the termination of life. Initial fever quite rare.
Skin pale, rarely ruddy. Skin of normal turgescence.	Skin usually of ruddy appearance, anemic only after profuse hemorrhages. Frequently the visible mucous membranes, and even the cheeks, slightly cyanotic. Patients sometimes present the chlorotic type.	Skin sallow, yellowish, dry, and flaccid. Marked cachexia.
Often conjoined with hysterical symptoms. Occurs at all ages, more frequently in women than in men.	Most frequent in middle life; rare in children. Accompanied by a varying psychical condition, frequently great depression.	Most frequent between the fortieth and sixieth years. Psychical condition that of depression; melancholia, but, strange to say, less profound than in severe cases of ulcer.

Nervous Gastralgia.	Ulcer.	Cancer.
No tumor on palpation, unless, as rare exceptions, when foreign bodies, hair, etc., have been swallowed. Chemism varies; absence of lactic acid.	When the ulcer is situated at the pylorus with consecutive hypertrophy, an ovoid, smooth tumor at the right of the median line may be palpated. Occasionally in old ulcers with a hard base and callous borders a palpable tumor with circumscribed encapsulation, perforation or adhesions with the head of the pancreas, the left lobe of the liver, the spleen or omentum, and does not move with the expiratory excursion. HCl present, and usually increased.	Tumor of varying size and shape, nodular or smooth, distinctly palpable: as a rule, passively moved, occasionally also in respiration. In the majority of cases no HCl; absence of pepsin digestion; lactic acid. Lab-ferment sometimes absent (cancer of the pylorus), sometimes present (cancer of the fundus). Secondary glandular enlargement. Metastases.
No symptom of perforation.	Perforation into neighboring organs with characteristic symptoms, frequently after apparent brief duration of the disease even occurring without prodromes.	Perforation; implication of neighboring organs only after prolonged existence of the disease.

Nevertheless, distinct as these three clinical pictures may appear to be upon paper, in practice the most prominent symptoms are often so ill-developed or so merged into one another that a precise diagnosis is impossible, as, for instance, in the onset of the ulcerative process. So long as the symptoms indicate only general digestive disturbances, so long as there are no typical gastralgic attacks, and especially so long as there is no trace of hematemesis, we have no clue by which to distinguish this condition from the great category of dyspepsias. An important aid in the recognition, which makes an early diagnosis possible, is the proof of hyperchlorhydria, although we should not forget that rare exceptions to this occur, and that chemically slight hemorrhages into the gastric and intestinal contents cannot be macroscopically recognized.

The diagnosis is most positive when we find the symptoms of typical gastralgia, vomiting of blood, blood in the feces, absence of tumor and cachexia. In such cases it is unnecessary to examine the patient with the stomach-tube; for this may be a serious procedure, and it had better be avoided. On the other hand, in indefinite cases the investigation of the chemism is absolutely necessary, but all precautions must be observed. The proof of hyperchlorhydria or of gastrosuccorrhea with a large amount of hydrochloric acid is then decisive, and indicates, particularly the latter occurrence, that the seat of the ulcer is at the pylorus,

and there is consequent stenosis. I have seen patients with undoubted ulcer of the stomach with extreme loss of strength, and, on the other hand, cases of gastric cancer with unimpaired strength, appetite, and the general habitus. Occasionally, as Leube also states, the diagnosis can be made solely by the efficacy or fruitlessness of a specific treatment for ulcer. Extreme difficulty in the differentiation may be caused by the above described tumor-like cicatrization, which draws the neighboring organs toward the base of an ulcer to which they are adherent, or which forms above a perforated ulcer. In the latter case the head of the pancreas, the left lobe of the liver, or, more rarely, the spleen, may be involved. In the gastrocolic ligament there is a lymph-gland, or a band of closely situated glands, which under some circumstances enlarge and become sensitive to pressure; they may be palpated as small tumors the size of a hazelnut at the lower boundary of the stomach, and have repeatedly caused me the greatest perplexity in diagnosis. In all of these cases the persistent size of the tumor, the maintenance of strength, and the presence of hydrochloric acid, favor ulcer in opposition to cancer, just as a course lasting more than three years and the absence of a typical cancerous cachexia favor the first named affection. Since we know that hyperacidity exists in numerous cases of ulcer, it is obvious that those distinct tumors of the stomach, particularly those situated about the pylorus, which in spite of typical signs of malignant cachexia run their course with profuse excretion of hydrochloric acid, may be referred to the fact that cancer has developed upon the foundation of a gastric ulcer. I have repeatedly observed such cases. In several instances in which a tumor of the pylorus while under observation and within a year developed to the size of a walnut, the acidity amounted to 104 and 101. Gastroenterostomy was performed, and at this time the tumor was inspected and subsequently removed; it proved to be an unquestioned carcinoma.

A test of the chemism, as advised by Gluczinsky, is valuable for the early recognition of beginning carcinomatous degeneration of an ulcer and may be carried out upon one and the same day, first upon an empty stomach, secondly, after a test breakfast, and, finally, after a midday meal. In a florid ulcer all tests give normal values; decrease or absence of one or more favors a beginning carcinoma. In 17 cases in my wards Dr. Sigel has put Gluczinsky's results to the test, and, although not in every case, in the main he confirmed them.

Diagnosis of Perforation.—The diagnosis of perforation of an ulcer is based upon the above described symptoms, and only those features will be discussed which may lead to error. Here we must consider perforation of a gastric carcinoma (a relatively rare occurrence), of the gall-bladder, that is, by a gall-stone, of the appendix, of an intestinal ulcer, and acute diffuse peritonitis caused by rupture of the spleen, of a pyosalpinx, or of an ovarian tumor. The greatest differentio-diagnostic perplexity may

arise from the rupture of a gastric cancer, which, however, as has already been mentioned, is extraordinarily rare because the neoplasm is prone to attach itself by inflammatory adhesions to the surrounding organs. The other possibilities enumerated may be excluded by a careful examination and the history. But there are other diseases which, without producing perforation and peritonitis, may simulate these conditions. Here are to be mentioned: Gall-stone and renal colics, torsion of pedicles, torsion of the ureters in movable kidney, severe gastralgia, poisoning, embolism and thrombosis of the mesenteric arteries, and, finally, even pleurisy and pneumonia, the latter affection occasionally, i. e., in a few cases, being complicated by a similar symptom-complex. I can only call attention to these occurrences; the diagnosis will be in part described in the following pages.

Unfortunately, we have no means of recognizing a threatening perforation because there is nothing to indicate whether, in a given case, we are dealing with a superficial or deeply invading ulcer. Symptoms persisting for a long time may, perhaps, be utilized in the latter sense, but the uncertainty of such a conclusion is obvious, and the great majority of cases of ulcer run their course for years without perforation.

The diagnosis may be very obscure when we are deciding between *cholelithiasis, renal colic,* and *gastralgia* occurring in the course of ulcer of the pylorus or ulcer of the duodenum; of course, not in typical cases of either disease. Recurring pain in the right hypochondrium independent of the ingestion of food, slight fever, jaundice, enlargement of the liver, pain over the liver, a palpable gall-bladder perhaps containing stones, drawing pains along the ureter, hematuria, the passage of gall-stones or renal stones are just as typical of gall-stone colic or renal colic as the total complex of the symptoms previously described is of ulcer. But in many cases the symptoms are so indefinite that confusion can scarcely be prevented. If in gall-stone colic jaundice is frequently absent or very feebly developed, there are, on the other hand, not seldom cases of gastralgia which run their course with mild jaundice, either because bile is forced into the blood-vessels from spasmodic contraction of the abdominal organs or because a rapid, transitory, sympathetic spasm of the hepatic duct occurs, and with this biliary stasis. The patients with gall-stone colic are apt to locate their pain in the median line, particularly women, in whom the topography of the liver has been changed from lacing. Fink even makes the very remarkable statement that among 403 cases of gall-stone colic which he observed in Carlsbad, in 380 = 94.3 per cent., gastric spasm occurred alone without pain in the hepatic region! If the pylorus is displaced somewhat to the right, or if the ulcer is situated in the horizontal axis of the duodenum, there can be no question of a local difference. Hence, it may for a long time, or perhaps always, be a mooted question whether cholelithiasis or gastralgia is present. But here hyperchlorhydria

of the gastric juice, if present, gives us a valuable clue. Acidity over 80, i. e., 0.3 per cent. HCl, may be utilized in this way.

A *tuberculous ulcer* is recognizable by its reaction to tuberculin, according to Petruschky's method. I should like to remark in this connection that we must never be content in such cases with one test if this be negative, but several injections, in increasing doses, must be given. Repeatedly I had no reaction with 1 and 2 mgm., but obtained a typical one after employing 3 and 5 mgm.

Position of the Tumor.—Only an unusually favorable combination of circumstances will enable us to recognize the position of an ulcer at the pylorus or in the duodenum, perhaps even in the greater curvature. On the other hand, by exclusion we may decide that the ulcer is situated elsewhere. An ulcer at the pylorus is characterized by sharp, localized pain a little to the right of the median line. Fleiner attaches great weight to the symptom of pyloric spasm. But the significant factor of pain cannot here be reckoned upon, and the assumption that ulcer situated in the cardia of the stomach is accompanied by sensations of pain immediately after eating while those at the pylorus produce pain only later has, I find, neither been sufficiently proven clinically, nor is it justified by the actual conditions. An attempt has been made to locate the seat of the ulcer from the position which some patients assume to alleviate their pain. If the pain is less in the left lateral position, the ulcer is said to be situated at the lesser curvature, or *vice versa*. This also is a very doubtful and uncertain symptom, inasmuch as it does not accord with the experience of the majority of patients. According to Gerhardt: "Sensitiveness to pressure and tumor favor the seat of the ulcer upon the anterior wall, pain in the back and hemorrhage its seat upon the posterior wall. The seat of the pain and its increase in the latter position often permit the differentiation of ulcer of the fundus or of the pyloric region. When an ulcer at the fundus is adherent to the spleen it may by producing splenitis give rise to chills, as I (Gerhardt) have seen in three cases." That gastrectasis indicates the seat of an ulcer at the pylorus or in the duodenum and contraction at the cardia, requires no special emphasis. When we reflect how vague is the symptom of pressure sensitiveness, how rare is the appearance of tumor due to ulcer in proportion to the total number of cases, how very difficult is it in these cases to determine the constriction of the stomach *intra vitam* since we do not inflate the organ or introduce the stomach-tube, and, lastly, when we remember that often several distinct ulcers are situated in different areas, no great weight will be attached to this symptom.

But, in my experience, the most reliable of the symptoms which have been mentioned is the pain, often spasmodic, in the region of the pylorus, therefore, in the right mammary line, localized below the border of the liver. As, however, the pylorus is occasionally very movable, and may

frequently be found even at the left of the median line, it is clear that pain in the latter region cannot always be referred to the greater curvature or even to the fundus. v. Leube very correctly remarks: "We must beware of such diagnoses. They are at least uncertain, as well as all diagnoses based upon the subjective symptom, pain, even when some special condition is pointed out by this; for instance, when pain is developed only on displacing the gastric contents from a particular position, therefore only appearing when the patient assumes the right lateral position." Nor must we forget the well known fact, to which Schütz has recently again called attention, that there are inflammatory conditions in the transverse colon associated with pain which may readily but erroneously be referred to the stomach.

It would materially aid us in the comprehension of this condition if we possessed a really good *gastroscope,* but, at present, this does not appear to be the case. It need hardly be stated that the same consideration which deters us from introducing a stomach-tube or a soft sound in ulcer is more strongly deterrent in the case of a rigid metal tube.

It is true that a stenotic gastric ulcer at the pylorus with consecutive gastric dilatation, and, *vice versa,* the same condition at the cardia with resultant contraction of the stomach, are readily recognized, the latter, perhaps, requiring the aid of the esophagoscope; but, even with this, we get little further knowledge, and to the cases mentioned by Gerhardt I may oppose another where the perforation of a broken-down carcinoma of the smaller curvature manifested itself by chills and intense pain, especially upon the left side. We are in an extremely difficult position when several ulcers are simultaneously present, and this condition is not rare. *Exact knowledge of the location is, therefore, for many and perhaps for most cases, very desirable,* and the more important because accurate location of the seat of the ulcer is of the utmost significance in the treatment, and particularly for operative intervention when hemorrhage occurs. How difficult this determination is may be understood from the fact that Schloffer was unable to find it in 2 of 5 cases even at the operation.

But even if we were so fortunate as almost to grasp the ulcer with our hands, i. e., to detect *tumor at the pylorus,* and although other factors, particularly the chemism of the stomach, the age of the patient, his general condition and strength, were of such a nature as to exclude carcinoma, an absolute diagnosis could not be made. Hence the following conditions come into view, and these we must discuss for a few moments:

I. *Pylorospasm.*

II. *Muscular hypertrophy,* that is, *cicatricial thickening of the pyloric region.*

III. *Carcinomatous neoplasm.*

Of the first it must be remarked that although its most frequent cause is an ulcer at or in the immediate vicinity of the pylorus, cases have been

undoubtedly observed in which pylorospasm occurs without any alteration in the mucous membrane that can be detected. Here the observations of surgeons are of great weight. Both Schloffer and Alberti have reported such cases.

The case of the first author was that of a woman, aged 44, in whom the gastric contents showed no hydrochloric acid, but lactic acid. At the operation an oval, slightly diagonal, extraordinarily hard, circumscribed resistance of about 2 cm. in length and 1 cm. in breadth, which prolonged investigation showed to be nowise altered, was found at the greatly narrowed pylorus, and on its posterior wall. The area in question was resected, and, most astonishing to relate, absolutely no pathological changes were found. Histological examination merely showed a slight hypertrophy of the musculature in this area, and Schloffer assumes the case to be one of circumscribed spasm of the musculature of the pylorus in its posterior wall.

The case of Alberti is interesting from the fact that at the laparotomy a pyloric tumor was found which was of uniform hardness, sharply demarcated toward the duodenum, but toward the stomach and particularly at its posterior wall it showed merely a coarse and firm wedge-shaped swelling amounting to about 4 cm. in length. Alberti looked upon it as a neoplasm, and concluded to perform pylorectomy, the more so since no adhesions or metastases were apparent. When, however, the lesser and greater omentum were detached, all signs of tumor suddenly disappeared, and the stomach, that is, the pylorus, became soft and perfectly normal. Pyloroplasty after Heinecke-Mikulicz was performed, and the pylorus was found to be narrowed but no firm cicatrix could be detected. The attacks of intense pain were arrested, and the patient, in spite of the continuance of symptoms of stenosis of the pylorus for five years, showed very slight dilatation of the stomach.

This case appears to leave no room for doubt. Here marked reaction for hydrochloric acid was always present. On the other hand, in the light of our experience in the Augusta Hospital, the case of Schloffer may be regarded as questionable.

We have observed two such cases in which resection was apparently performed on account of a benign pyloric tumor, and the most minute histologic examination of the resected tissue not only by us but also by our pathologists showed no change characteristic of carcinoma. The first case was that of a woman, aged 47, who had been suffering for several months from a gastric affection, and complaining of vomiting and eructation; examination showed a moderate gastric dilatation and a pyloric tumor causing stenosis. Free HCl was repeatedly found to amount to 0.5–0.6 per cent., albumin digestion to about 75 per cent. Lactic acid and blood were never present. The gastric tumor was resected and gastroenterostomy performed. In 20 sections from the resected tissue nothing

characteristic of cancer could be found. For five years the woman remained in good condition, then ascites developed combined with abdominal symptoms which pointed to a tumor of the abdomen. An indistinct resistance could be palpated both by the vagina and by the rectum.

Operation performed in the summer of 1901 revealed extrauterine pregnancy and, simultaneously, a neoplasm of both ovaries which, at the histologic examination—the patient died soon after the operation—proved to be an adenocarcinoma. There can be no doubt that this was a metastasis from the stomach.

The other case occurred in a man, aged 37, who, six weeks prior to Christmas of 1897, was suddenly attacked by diarrhea which continued until the time of his admission upon the 9th of March, 1898. Fourteen days previously he began to vomit brownish masses, but had never suffered from pain in the stomach; he felt quite strong, and had a good appetite. After inflation of the stomach a tumor the size of a small apple was found in the epigastric region upon the right below the border of the liver. The stomach was slightly enlarged; no glandular enlargement. Acidity was 58 upon the empty stomach and lactic acid 25. In the test breakfast, acidity was 54, lactic acid 16 (0.058 per cent.). Pepsin digestion was barely 10 per cent. Laparotomy upon the 14th of May disclosed a tumor of the greater curvature extending to the pylorus, and in its middle a deep ulcer. About two-thirds of the stomach was resected, and on the remainder of the stomach gastroenterostomy antecol. anter. was performed. The patient was discharged cured upon the 7th of April.

Although the diagnosis of *carcinomatous ulcer* was made from the macroscopic appearances, histologic examination did not reveal a characteristic picture. The man has just been readmitted to my ward with a large, undoubtedly malignant tumor of the greater curvature.

We see, therefore, that in the differentiation of these so-called spastic tumors and muscular hypertrophies of the pylorus we must be very cautious. It is not surprising that, in spite of resection of an apparently healthy organ, metastases should develop. Years ago I stated that typical, cancerous nests might be found in the submucosa and muscularis far beyond the macroscopic, visible portions of a malignant tumor.

Tumors due to muscular hypertrophy are generally differentiated from spasm by their consistence. The same cause which generates them always produces stenosis of the pylorus, whether they originate from the cicatrix of an ulcer causing stricture, or from reflex contraction, from the irritation of a fresh ulcer by the ingesta, or, finally, from a malignant neoplasm. I must also mention that occasionally a gall-stone is incarcerated in the pylorus (Naunyn, Elsner) or tumors from hair or trichobezoar may be present. In how far dilatations of the stomach occur in connection with these conditions, and how far they may be attributed to it, depends upon the duration of the affection, upon the permeability of the stenosis, and

upon the degree of the compensatory muscular hypertrophy of the stomach. Here, however, there are marked gradations in the condition. We meet with convincing proof of the fact that ulcer of the pylorus frequently merges into carcinoma of the pylorus and here, as in other areas, persistent mechanical irritation from the chyle which presses through the pylorus performs an active part. These are the cases in which hydrochloric acid secretion and the peptic function of the stomach are long retained, although they finally decrease from the norm. With these also belong some of the cases which I first mentioned, cases in which, on account of the chemism of the stomach, the diagnosis of a benign tumor of the pylorus was made, while operation or autopsy revealed that we were dealing with a more or less carcinomatously degenerated old ulcer. It always appears to me that the general condition of such patients suffers but little provided high-graded pyloric stenosis does not occur, and this is another factor which may lead to diagnostic errors.

In conclusion we must consider the last of the three groups mentioned, namely, *carcinomatous tumors situated at the pylorus or in other portions of the wall of the stomach.* Here, of course, we are only dealing with those cases in which the age, the patient's strength, the chemism, the gastric contents, and the objective condition raise doubts as to whether we have before us a carcinoma, the old cicatrix of an ulcer, or an epigastric process.

In my opinion, during the short or long course of the disease a certain grouping of the symptoms may sometimes entirely preclude our making a diagnosis at the time, because neither the individual symptoms nor their totality are sufficiently clear to lead in any direction to a decision. This is particularly true of two symptoms to which we otherwise, quite justly, attach great significance: The *pain* and the *emaciation.*

Here I entirely leave out of consideration those cases in which gastralgia occurs at the initial stage of pulmonary phthisis in young, chlorotic and anemic persons or where these attacks occur in the so-called preataxic stage of tabes dorsalis (locomotor ataxia), because these may be easily differentiated by a minute examination which, in women, should include also the genitalia.

A number of years ago Aufrecht called attention to cases of severe gastralgia without anatomical findings which even showed a family relationship. I have never seen cases of this kind, and must say that Aufrecht's brief report does not appear to me convincing: "At the autopsy nothing was found in the stomach, the biliary passages, or the duodenum to make clear the cause of the disease."

In old gastric ulcers the *typical pain* beginning at a definite time after the ingestion of food is either absent, or it is so irregular that it does not differ from cardialgia due to other causes; therefore, it does not differ from the pain produced by perigastric adhesions and carcinoma,

either typically, or by its seat, or by the frequency of its appearance. Here we must also consider the typical pains due to a *hernia of the linea alba.* In general these may be readily demonstrated, but it is necessary to bear them in mind, for, in my experience, they lie somewhat beyond the pale of the experience of most practitioners. By proving that the condition was hernia, and by a slight operation, I have succeeded in promptly curing many a case diagnosticated as gastric ulcer.

If, however, there are no well developed attacks of pain, and we are dealing only with more or less decided discomfort, a sense of pressure, and fulness after eating, we know that these may exist for a long time and, even until death, be the only symptoms in patients with cancer.

It is true that in ulcer emaciation is usually much less marked than in cancer, and that the color of the skin characteristic of malignant cachexia is usually absent. Where, however, an ulcer forms in debilitated, nervous, hysterical persons or, *vice versa,* in the robust who, from a fear of pain, limit for a long time the amount of their food, rapid and striking losses of weight occasionally occur and lead us to consider seriously the existence of carcinoma.

But the condition of the peripheral lymph-glands should give us reliable information on this point!

Long ago, in 1886, Dietrich proved, after a careful examination of normal persons, that is, those not suffering from malignant disease, the great frequency of slight glandular enlargement—thus, for example, the inguinal glands in 92 per cent. and the axillary glands in 64.9 per cent. were swollen to the size of a bean. But decided tumefaction must be present for this to be of any importance. Tarchetti lays special stress upon enlargement of the supraclavicular glands which he met with in 18.4 per cent. of cases of cancer. But in my experience statistics of glandular enlargements *are not very reliable.* In 125 cases of positive ulcer of which I have notes regarding this, sixty times the inguinal glands were enlarged to the size of a bean, and twenty-four times the glands of the axillary cavity to the size of a pea. Nevertheless, I must admit that the condition of the lymph-glands, particularly the enlargement of the left-sided supraclavicular glands, is of some significance. Still greater weight must be attached to the state of the *tongue,* which in ulcer—especially in recent ulcer—is red, moist, glistening, while in cancer it has a white coating and is usually dry, in nervous disturbances it is deeply fissured, particularly upon the sides, so that the tongue resembles a freshly ploughed field, or, to use a more striking comparison, a glacier with its clefts. And this, fortunately, again brings us to the symptom *per tot discrimina rerum* to which the ancients, without any knowledge of our modern methods, attached so high a diagnostic value!

Duodenal Ulcer.—What is true of the seat of ulcer of the stomach is also true of the seat of ulcer of the duodenum: in at least 90 per cent.

of the cases it is impossible to decide positively whether we are dealing with ulcer of the duodenum or ulcer of the stomach. The duodenum, and particularly its horizontal branch, so far as these conditions are involved, is only to be regarded as an appendage to, or a portion of, the stomach, and ulcerative processes in one, as in the other, produce the same symptoms. Factors which favor ulcer of the pylorus are also operative for ulcer of the duodenum, all the more so as the ulcer sometimes directly attacks the duodenum from the pylorus. A *duodenal ulcer* is likely when the pains only set in some time after the ingestion of food; their seat, as well as a passive sensitiveness to pressure, is decidedly to the right of the parasternal line, and profuse hemorrhagic dejecta and hematemesis may appear. Jaundice and peritonitis have been repeatedly observed. The fact that ulcer of the duodenum frequently occurs in old persons after extensive cutaneous burns may in a given case be of value in the diagnosis. A point of support, but no more than this, is the rare occurrence of duodenal ulcer, for, according to Williglk, to 225 cases of gastric ulcer there are only 6 ulcers of the duodenum, according to Trier only 28 to 261. But this small percentage includes a number of cases in which ulcer was present in the stomach and duodenum simultaneously. Gastralgia is said to be not so frequent as Budd believes, because the duodenum is exposed to less distention and less change of position than the stomach. Jaundice, which is very rare in ulcer of the duodenum, can be little utilized in the diagnosis from the circumstance that, in the main, intestinal hemorrhage is more frequent and hematemesis rarer, but gastric ulcer also leads to hemorrhage from the intestines, and ulcer of the duodenum may be accompanied by hematemesis.

PROGNOSIS

Until recently, and quite properly, a doubtful prognosis was given in gastric ulcer when it was differentiated by the signs which have been mentioned. Since we have become able to make an early diagnosis, and to separate it from the forms of dyspepsia, since the principles of treatment have been clearly established, and we are in a position to use them at the onset of the process, the prognosis in at least the early stage of ulcer has decidedly improved. If the patient is subjected to rational treatment at the proper time, i. e., if a rest cure is instituted, well founded hopes of recovery may be entertained, and even in classical ulcer recovery or decided improvement may be looked for. Unfortunately, during the first stages, which subjectively do not occasion great difficulty, very few patients are inclined, or are in a position, to submit to such treatment. If, however, we succeed in curing the ulcer by permanently changing the abnormal composition of the blood or the secretion of the gastric juice, the fear of relapse is also removed, which, otherwise, always threatens

and only too frequently occurs. Invariably, however, and particularly in the healing of extensive ulcers, there is danger of a permanent damage to the health from the consequences of cicatricial tissue formation, and this cannot be lost sight of. In such cases the prognosis must be made with great caution. That it is not bad is, nevertheless, evident from the well known fact that cicatrices from gastric ulcers are found about twice as frequently as open ulcers. In hemorrhage, provided it is not immediately fatal, the prognosis is, as a rule, favorable, and it is better the younger the individuals in question. By suitable treatment we usually succeed in mastering the hemorrhage, and even extreme anemias are improved in a relatively short time. At the Surgical Congress in Berlin (1897), v. Leube gave the following statistics of his cases: Of 424 hospital patients 314, = 74 per cent., were cured after a treatment of four weeks, 93, = 32 per cent., were improved, 10, = 2.4 per cent., died, 7, = 1.6 per cent., were unimproved. Therefore, in only 4 per cent. was careful treatment without result. This coincides with my reports of 233 hospital patients, in which I had 76 per cent. of recoveries.

But it cannot be denied that in these statistics the factors, which have often been mentioned, of unlike circumstances and insufficiently long observation play a part. The surgeon reaches other results than the physician, and thus it comes to pass that v. Mikulicz gives a mortality of from 20 to 30 per cent., while Leube has only 4.1 per cent. It is obvious that the brief period of observation in the hospital can only be regarded as corresponding to the "healing" of the ulcer; it does not strictly apply to the period after the patient's discharge from the hospital, and tells us nothing of the mortality. The latter depends upon accidental conditions, and can only be arrived at with some degree of certainty from the reports of many autopsies. But the "statistics of cures" are also open to doubt from the fact that groups denominated as ulcer include many cases in which true ulcer was not present, as is evident from the nature of these cases. For this reason I have refused to utilize my statistics in this way. J. Schulz has decidedly cleared the situation. He analyzed 291 cases, 184 from the Breslau Clinic, and 107 from the Eppendorf Hospital, all of which were recognized as ulcer by the appearance of hemorrhage. Of course under these circumstances a diagnostic error was not absolutely excluded, but, nevertheless, it was scarcely possible. Questions were sent to all of these patients from which deductions were to be made regarding the results of treatment. One hundred and fifty-seven answers were received, and the accurate investigation of these justified the conclusion that internal treatment of ulcer of the stomach according to the method instituted by v. Ziemssen and Leube gives a permanently good result in 64 per cent. In 18 per cent. relapses occurred, but, nevertheless, most of these patients were finally cured. It might be assumed that under renewed proper treatment the others would decidedly

improve. In 18 per cent. the treatment was without result, and of these 7.6 per cent. died. If, therefore, we count those who were not benefited among the unsuccessful cases, 64 per cent. were cured, in 13 per cent. there was temporarily either no result or a relapse, and in 23 per cent. absolute failure.

TREATMENT

Whenever possible, gastric ulcer should be treated by the rest cure inaugurated by v. Ziemssen and v. Leube, in which all irritation of the stomach is to be prevented, just as a fractured bone is immobilized by a plaster dressing, naturally with the difference that in the latter case this rest is absolute while in the former it is only approximate. Rest in bed and nutrition by the rectum or by food which burdens the stomach as little as possible are the foundations of this treatment which, in England, was long practised by Wilson Fox and Balthazar Forster. As adjuvant, anodyne remedies, and at the same time calculated to lessen irritation, moist heat in the form of hot compresses and the drinking of hot Carlsbad water or a solution of Carlsbad salt which neutralizes acid, are recommended. v. Leube administers the Carlsbad water lukewarm; the compresses, however, should be as hot as they can be borne; according to his latest reports, he no longer uses nutritive enemata. To the latter, however, I attach the greatest importance, while I have discarded the hot compresses, chiefly because they leave an ugly pigmentation upon the abdomen. It is astonishing how well the majority of patients will bear exclusive rectal alimentation for three, four or more days, the nutritive property of which, according to all recent investigations (Eichhorst, Ewald, Huber, Rost and others), and in spite of several attempts to depreciate its practical value (Plantenga, v. Mering), admits of no question, particularly if small quantities of a 5 per cent. solution of cocain upon pellets of ice be given by the mouth.

Whether nutritive enemata, as Zierko maintains, actually diminish the acidity of the gastric juice, has not yet been positively determined.

Acting upon Kussmaul's advice, I have for some time administered large doses of bismuth in suspension (about 15 to 20 grams in 200 of water daily, divided into three doses, well shaken, and given before meals) which, according to the experimental reports of Matthes, forms a protective coat over the exposed, ulcerated surface.

This treatment, according to universal experience, gives excellent results where the ulcers do not invade too deeply, or where the condition resembles that of a florid ulcer, while, in truth, other affections—perigastric adhesions, neuroses, cholelithiasis, renal stones and the like—are the causative agents. Bourget has lately declared that rectal alimentation is rather harmful than beneficial (why?); on the other hand, he administers rice soups and milk and rice, and, therapeutically, washes the stomach

with a 2 per cent. solution of iron chlorid, to which ½ per cent. of potassium chlorate is added; so far, I have had no occasion to test his method.

Leube attaches weight to the fact that Carlsbad salt has a neutralizing, and, on account of the sodium chlorid it contains, also a stimulating effect; but the latter might rather be looked upon as deleterious, since we know that in many cases the acidity is greatly increased, and therefore a depressant rather than a stimulant is indicated. As to the neutralization, or decrease, of the acidity, I do not attach much importance to this if it is observed but a single time in an empty stomach, provided there is not always a secretion in the stomach when it should be empty. On the contrary, the diminution of the hypersecretion and the laxative effect of the neutral salts, as well as the soothing influence of large quantities of warm water, appear to me to be of the utmost importance. If the water of simple alkaline springs has been found to be less effective than that of the saline alkaline springs, it is only because we have forgotten to produce the desired laxative effect by other means.

Where there is no laxative effect from Carlsbad water, as is frequently the case, it must be brought about by the addition of Glauber salt or, still better, by vegetable laxatives, preferably rhubarb or senna. It is unnecessary to adhere rigidly to a formula, the principle alone is important. Whether the pains are relieved by warm fomentations or, in case these are ineffectual, by small doses or subcutaneous injections of morphin, whether the patient is given a solution of Sprudel salt or the natural spring water from Carlsbad, or, for example, Ems, Vichy, or the Neuenahr Spring, and the absent neutral salt be supplied by the addition of other aperients, is immaterial. Of the Carlsbad spring water 300 to 500 c.c. should be given. But which spring? This is unessential since there are no important differences in the chemical combination, and the differences in temperature of the individual Carlsbad springs are of little consequence because these waters can only be taken as hot as the patient can bear them; therefore are all taken quite hot. Fifteen grams (about one tablespoonful) of the salts should be taken daily, small quantities dissolved in half a liter of water at intervals, with corresponding pauses between. For the first three days absolutely no food is to be administered by mouth, but a nutritive enema is given three times daily; subsequently, besides the enemata, milk, or milk in flour soup, in tablespoonful doses, or bland pigeon or chicken broth. The milk, on account of its fine floccular coagulation, has some pepsin added. If this diet is well borne, it is added to in a manner soon to be described; otherwise, absolute rectal nutrition is again instituted. If no pain follows the careful administration of milk, we may permit somewhat larger quantities (up to a flat plateful, i. e., about 180 c.c.), leguminous flour soup, then legumes, later pappy food made of chestnuts, sago, tapioca, Kufeke's flour, hygiama and others, and later small quantities of meat. Among nutritive sub-

stances cow's milk takes the first place; it was first advised for this purpose by Cruveilhier. It is suitable because it contains all of the nutritive elements in solution, that is, finely divided, is free from irritating substances, because the acid is neutralized, and because the coagula which forms from the action of the gastric juice remains soft. The patient, however, must drink it very slowly and lukewarm. To prevent the floceulent coagulation of the milk, and the irritation of the ulcerative surfaces due to this, I now add pegnin (lab-ferment), which produces a very fine flocculent coagulation. v. Mering advises lab cheese for patients who cannot take milk. Besides pigeon or veal soups, the yolk of an egg, and beaten-up egg albumin, pulverized meat or leguminous soups may, perhaps, be given. We must limit ourselves to these foods until the severe symptoms have disappeared. In the third week a food richer than this, both quantitatively and qualitatively, is permissible, and we should then carefully try food of somewhat greater consistence, such as scraped raw ham, raw or very soft boiled eggs, scraped venison or the breast of fowl, rolls or zwieback softened in cocoa, but milk is always preferable, and we should always be ready to return to a simpler diet as soon as symptoms, or even pains, appear.

Even small portions of coarse bread, legumes, fruit, cabbage, salad, pickles, mushrooms, high spices, fatty foods or those prepared with vinegar, liver, fatty acids, confectionery, alcohol and coffee are to be strictly prohibited.

In regard to the *diet in hyperchlorhydria,* the article by Strauss, "The Diagnostic and Therapeutic Significance of Secretory Disturbances of the Stomach" (see this volume), which embodies the latest views on this subject, should be consulted.

It is absolutely necessary that the patient take but little food at a time but somewhat more frequent meals, that he eat slowly, avoiding all hot food; after recovery he should never overload the stomach, but must refrain from foods difficult of digestion or highly spiced, so as to prevent any lesion of the cicatrized area.

Lenhartz proposes quite a different dietetic treatment. Starting from the fact that hyperchlorhydria, chlorosis and anemia frequently develop in the course of ulcer, he permits his patients from the start to take concentrated foods rich in albumin. The patients, even when hemorrhage has occurred immediately before treatment, receive doses from a spoonful up to 300 c.c. of iced milk in which as many as 3 eggs have been beaten; from the third day sugar is allowed, and from the sixth day scraped meat, then milk, rice, and fine bread; from the tenth day raw ham and butter. Rest in bed, applications of ice, one to two grams of bismuth daily and, perhaps, iron with arsenic are ordered. In sixty cases treated in this way the results are said to have been very good; one case proved fatal, seven cases of relapsing hematemesis remained in the hospital.

The pains ceased almost immediately after the ingestion of food rich in albumin, and the patients recovered more rapidly than those on the preceding diet. Of 25 patients who were subsequently questioned, 18 (72 per cent.) were entirely free from symptoms.

The editor of the *Fortschritte der Medicin* quite properly remarks of these reports that the results are no better than with the old, reliable diet, and that there are very few who would risk the employment of such food in the stomach of a patient with ulcer. The reason given by Lenhartz-Wagner in favor of this coarse diet, that the patients recover more quickly, does not appear to me to be justified. Patients with ulcer recover very rapidly when the pains cease, and this takes place the more quickly when the gastric mucous membrane is allowed absolute rest instead of being stimulated to greater activity.

It is true that patients on this diet are at first debilitated and lose weight, but even here we must individualize. To attempt to regulate the diet in these cases according to calories is a form of play under a scientific cloak which to-day is much in vogue. As soon as the patients are able to eat at all they are prone to eat so much, even without calory force, that they soon recover from the former undernutrition. Convalescence is rapid, gastralgia ceases, and the time arrives when we must consider the second indication, the strengthening of the constitution.

For this purpose *iron preparations,* either alone or in combination with arsenic, are serviceable, the former in cases of pure chlorosis or anemia, the latter when we are combating a weakened nervous system, and it is desirable indirectly to influence this by the direct stimulation of metabolism. The old opposition to the employment of iron in ulcer of the stomach was founded on the experience that iron is frequently badly borne while the florid process is present; this, however, does not hold good when improvement sets in. Which iron preparation is to be employed depends largely upon personal preference; each day brings forth a new one, and in one case this, in another that, is the better tolerated. Of late, I have frequently employed triferrin which, without exception, was well borne. I formerly used arsenic in the form of Fowler's solution with tinctura ferri chloridi. After Liebreich's investigations arsenious acid appeared to be more serviceable and I now employ this in pill form, 1.5–2 mgm. of arsenious acid, 2 cgm. of ferri sesquichlorat. Considerably smaller but decidedly active doses of iron and arsenic, as has been proven by investigations in metabolism conducted by Ewald and Dronke, are given in the waters of Levico and Roncegno and the Guber Spring, which are excellently borne. The remedy should be given in increasing doses and after meals. This régime must be continued for months, and then the administration of arsenic must be interrupted for three to five days about every three weeks. The diet may gradually become more liberal but, nevertheless, must be strictly regulated for months, and patients who show

a tendency to exceed their allowance must be given a written diet list indicating the amount of food permitted.

It is sometimes impossible to carry out the rest cure mentioned above because there are many patients who are unwilling, or not in a position, to undertake it, and in many cases it is necessary to fulfil a stringent *indicatio symptomatica.*

Under these circumstances treatment with large doses of bismuth is recommended. Kussmaul (Fleiner) advised that this in suspension be introduced in large doses into the stomach previously washed out, and that the patient then, according to the suspected seat of the ulcer, assume for some minutes such a position as will enable the drug to sink to the lowest area; therefore, for example, in ulcer of the pylorus the right lateral position. Since, as a rule, I avoid lavage in ulcer, and because I know that the stomach is usually empty early in the morning, and also that by a previous drinking of water the gastric juice present may be diluted and more rapidly propelled into the intestine, and since, moreover, it is scarcely possible so to cleanse the gastric mucous membrane as to remove particles which may possibly be adherent to the ulcerated surface, and this, at all events, would necessitate the use of many liters of water, therefore I omit the washing and permit the patient to drink the bismuth suspension upon an empty stomach. The beneficial results I have obtained prove the reliability of this modified process. Indeed, the patient's tolerance of the drug and the ensuing freedom from pain are quite remarkable, although not so invariable nor so prompt as would appear from Fleiner's report. This is quite natural; the conditions are occasionally much more complicated than we assume and the method presupposes to be the case.

The dose is large, apparently unlimited, and may be given for an indefinitely long period. In one of my cases the patient received over 800 grams in the course of a few weeks without the slightest inconvenience, not even constipation resulting. In this case, however, considering the size of the dose, the result was not very satisfactory. The same may probably be inferred from the large quantities employed by Fleiner (up to 1,000 grams). In place of bismuth, Pariser advises equal parts of the cheaper white chalk and talcum, one to one and a half teaspoonfuls in water in the morning upon an empty stomach, and in the evening three hours after the full evening meal. Under this treatment the feces remain light in color, and small hemorrhages are much more readily recognizable than in the dark bismuth feces. In place of bismuth subnitrate, bismuth subcarbonate (Boas) and bismutose have been advised.

If all proceeds as we anticipate, and a protective covering of bismuth forms over the ulcer, this has not only a symptomatic but a curative effect. Under this protection the ulcer has time to heal, and, as Matthes has shown, the formation of granulation tissue at the base of the ulcer and the proliferation of glandular and other epithelia take place.

Under thorough treatment which is long enough continued, in my experience for at least four weeks, recent ulcers promptly heal. In some cases a single treatment is not sufficient, but a repetition is necessary. The result of this treatment, however, is generally so uncertain that with an unsuccessful issue we may at once assume either that an incorrect diagnosis has been made (particularly the pure neuroses lead to errors) or that we have old, deeply invading ulcers with broken down, excavated borders, that malignant degeneration has begun or induration has developed. In such cases other curative measures are indicated,—gastric lavage, tonics, and stomachics, small doses of mild narcotics, aperients, and the like. The indications for the employment of these remedies must be obtained by testing the functional activity of the stomach.

Of additional remedies silver nitrate has also been advised, best in solution, and in decided doses (0.2 silver nitrate to 200.0 of water, a tablespoonful every two hours). With this remedy I have sometimes seen decided amelioration of the symptoms, and even a complete cure; in other instances, after a little time the remedy had to be stopped because increased disturbance of the stomach, nausea, anorexia, coated tongue, and derangement of the intestinal activity, diarrhea or constipation, appeared. In one of my cases, every time a spoonful of the silver solution was taken watery dejecta accompanied by severe abdominal pain followed.

In my opinion the dietetic principles enunciated above are also important in the treatment of ambulatory cases, and the diet should, at least, be followed as far as possible. As the patients digest meat better than starches and vegetables, they instinctively eat less of the latter, and eventually, therefore, suffer from a monotonous meat nutrition, i. e., they emaciate, and become nervous and irritable. This must be counteracted as much as possible, and larger quantities of fat in the nourishment are not contraindicated because, as I showed some years ago, fat has the effect of decreasing acidity. I endeavor to modify the hyperacid gastric juice by repeated small doses of alkali combined with rhubarb and cane sugar or milk sugar. Rhubarb has a mild action upon the intestines, the sugar has a decided anodyne effect, and for this purpose has been repeatedly advised. I use a powder of about the following composition:

℞ Magnes. ust., Natr. carbon., Kalii carbonic., āā 5.0
Pulv. rad. Rhei 10.0
Sacch. lactis 25.0
M. D. S.: Every hour enough to cover the tip of a knife.

This is to be taken dry; I have seen good results from its use. Patients who know by experience the beneficial effects of alkalies, particularly of

soda, are usually afraid of taking too much. In this respect their minds may be easy. I have never yet observed a deleterious effect from the too long-continued use of an alkali, particularly of sodium bicarbonate.

For the catarrh which accompanies ulcer, Ord advises potassium iodid with the addition of sodium bicarbonate in about the following formula:

℞ Kalii iodat..... 2.0
Natrii bicarbonic..... 5.0
Acid. hydrocyan. dil. (m. 2 per cent. acid) gutt. tres
Inf. rad. Gentian.....3.0: 150.0
M. D. S.: A tablespoonful three times a day.

It is well to remember the advice of Pariser that women who have suffered from ulcer, even after treatment is discontinued, should be kept in bed during the next two or three menstrual periods and on almost the same diet as while suffering from ulcer.

For the severe gastralgia, morphin internally or subcutaneously ranks first. Chloroform solutions (1–120, a tablespoonful every two hours) or chloroform in drops (5 to 6 in a teaspoonful of water or on a pellet of ice) occasionally produce excellent effect, not only upon the momentary pain but upon the course of the process in general. For a long time I have been in the habit of administering bismuth in suspension with a one per cent. addition of chloroform, or, instead of using distilled water, I employ chloroform water. Tincture of iodin, 5 drops, three times daily in water, occasionally acts as an anodyne. Of other sedatives I have occasionally employed lupulin, extract of cannabis indica, extract of hyoscyamus, and extract of belladonna, but have always been compelled to return to morphin or codein. Cannabis indica, in particular, which is so greatly praised and was advised by G. Sée, has in my hands repeatedly failed to show any anodyne or quieting effect, but, on the contrary, has produced an unpleasant irritation. Formerly leeches were applied at the seat of the affection, and also blistering plaster; it was painted with strong solutions of iodin, and even the actual cautery was used. We are content to-day with the ice bag, or the application of ice-cold or warm compresses, or Leiter's coil which, where the circumstances permit, is the most cleanly and convenient mode of employing cold.

Treatment with Oil.—Treatment with *oil* (linseed oil or olive oil) has been praised by various authors. Cohnheim, in 1889 and later, was the first to report surprisingly good results. The oil is to be given in gradually increasing doses from a tablespoonful to a wineglassful or may be poured in through a stomach-tube. Agéron advises the admixture of 10 grams of dermatol with 200 of the finest linseed oil or poppy seed oil. Walko adds bismutose or bismuth. The oil is best taken while the patient is in the recumbent posture, and Agéron has the patient assume the dorsal

decubitus, raising the pelvis after taking the remedy to relieve the greater curvature. Aside from the fact that the stomach-tube should not be introduced in cases of ulcer without very clear indications—which I think do not include the pouring in of oil—I have previously cited a case in which the oil caused such great nausea and retching that it was the immediate cause of a severe gastric hemorrhage. Cohnheim, however, in about 30 cases, even with irreparable organic changes at the pylorus as well as in functional disturbances, has seen no unpleasant secondary effects but usually a very favorable influence upon the pain, the spasm, the general nutritive condition and the degree of acidity of the stomach. Even cases in which operative interference had been advised by others were cured by this treatment. According to their nature, cases of the first category can only be symptomatically influenced, while permanent results have actually been attained in spastic stenosis of the pylorus, in ulcer, in fissures (?), in erosions of the pylorus, in pyrosis hydrochlorica with acid gastritis, and similar conditions when a pure neurosis formed the foundation of the difficulties, and provided no complicating perigastric processes existed. Instead of oil, an emulsion of almond milk (about a tablespoonful of sweet almonds ground up in about 200 of water) also has a quieting, but not a nutritive, effect. The oil has the following merits: It quiets spasm, it lessens friction, it decreases the acid secretion, and it assists in the nutrition.

Cohnheim, in his last publication (1904), remarks that it is strange that his reports have been confirmed only by Walko. This may be for two reasons. Either the process has been generally satisfactory, and only by accident others have failed to praise it, or the results lauded by Cohnheim have not been attained by others, and judgment has been suspended. The latter describes my own position. Ever since Cohnheim's first publication I have repeatedly employed the oil treatment in my private practice, in the Hospital, and in the Clinic, and I have observed some beneficial, and also some very ill, effects. The oil was so repugnant to many of the patients that it was impossible for them to take it, and in others it subsequently caused complete loss of appetite, eructations and vomiting, no matter whether given by the mouth or introduced through the stomach-tube. Fischl in a study of 19 cases arrived at the same conclusion. As the poor results were observed chiefly during the first trial, I must acknowledge that no extensive test was made by me, so that a definite statement regarding the method is at this time impossible. Cohnheim's last publication may induce me methodically to test the oil treatment anew. But at present, on the basis of my previous experience, I must maintain that in this treatment we possess no panacea for the symptoms in question.

Xeroform, one of the newer remedies (0.5 gram four times daily) has been much praised. Jaworski advises, under the name of aq. alcalina effervescens, a solution of sodium bicarbonate (8 or 5), sodium salicylate

(2½ or 2), and sodium biborate (2 or 1) in a liter of water, one-third to one-half of a tumblerful several times daily.

For the vomiting nothing is better than a carefully regulated diet. Large quantities of hot water may be taken several times daily, also pellets of ice with chloroform. Tincture of iodin (15 drops in 150 of water) has been advised by some authors. Special care is necessary if *hematemesis* appear; when very profuse this is self-evident, but it is also enjoined when smaller hemorrhages take place. Under all circumstances, the first requirements are absolute bodily and mental rest and the avoidance of all internal and external irritation of the stomach. Even in smaller hemorrhages, if circumstances permit, the patient should be restricted to this régime for several days, and the fullest precautions should be taken because these small hemorrhages are very frequently only the precursors of more profuse ones. Small pellets of ice, ice-cold tea, or ice-cold solutions of peptone in spoonful doses may be given. I do not give milk in such cases unless I know that the patient takes it well, but for the first day order either a solution of grape sugar to which some meat peptone bouillon is added and given ice-cold, or I administer cold, gelatinous soups of barley or oatmeal gruel; where the circumstances permit, nutritive enemata are cautiously given. The fluid extract of ergot, 2.5 to 5 of water and glycerin in equal parts, is subcutaneously injected in the gastric region, one to two syringefuls several times daily, but it must be remarked that ergotin in some persons gives rise to very unpleasant symptoms of constriction and vertigo. I have been unable to convince myself of the reliability of the fluid extracts of *hydrastis canadensis* and *hamamelis virginica,* which have lately been much used. When there is great irritability of the stomach, the injections of ergot should be combined with injections of morphin. This will usually arrest hemorrhage, provided large vessels are not implicated. Formerly lead acetate, iron chlorid, and oil of turpentine were employed internally because of their presumable styptic influence, but these are no longer used because we possess a more rational and active remedy in ergot. In some instances in which hemorrhages recurred for several days and the above measures were futile, the bleeding ceased upon *lavage of the stomach with ice water.* After preceding cocainization and the injection of a small quantity of morphin, the stomach-tube was carefully introduced, and the stomach wall repeatedly sprinkled with ice water, large amounts of hemorrhagic gastric contents being first washed out, whereupon the hemorrhage immediately ceased. Before proceeding to operate in such cases, this maneuver should always be first tried. It is occasionally astonishing to see what large quantities of hemorrhagic fluid are evacuated from the stomach. As some blood usually passes into the intestine and is there decomposed, and perhaps may set up irritative phenomena, it is advisable, if there is no spontaneous passage, to employ mild evacuants, preferably rhubarb with sulphur, or enemata; if symptoms

of collapse appear, injections of camphorated oil (1:6), enemata of wine, or wine with egg or peptone, and hot applications to the extremities. With threatening hemorrhage, a very small pulse, anemic murmurs over the heart, or cerebral anemia, normal saline infusions are indicated. Subcutaneous infusions are best given by employing a rather large Pravaz cannula. If the salt solution (7.5:1,000) warmed to the temperature of the body is injected simultaneously through two needles, and the fluid introduced is disseminated by gentle friction, one liter of water may be introduced in a very short time. We prefer the subclavicular region as the point in which the needle should be introduced. In favorable cases the blood is rapidly regenerated. In a patient, aged 25, who had received an infusion, the blood upon the next day showed 2,100,000 erythrocytes, two weeks later 3,560,000, with a slight increase of the leukocytes.

Adrenalin or suprarenal extract has also been advised for gastric hemorrhage. Fenwick employs 1.3 grams of the dry glandular substance boiled in 230 of water. Internally or subcutaneously it is more rational to employ adrenalin hydrochlorid in solutions of 1:1,000, 20 to 30 drops internally three to four times daily, subcutaneously 0.5–1 c.c. several times daily. There are quite a number of reports (Roussel, Renon and Louste, Mills, Kirch, Mamlock and others) in praise of its action, and authors are at least unanimous on the point that no unpleasant sequelæ (glycosuria, marked increase of blood pressure) appear even after its use for weeks.

Up to the present, I have employed the remedy internally in two cases, and it failed entirely. I must also say the same of *gelatin,* of which I gave a 10 per cent. solution up to 100 c.c. by mouth twice daily (it may also be used by enemata). In one case the hemorrhage continued, in two other cases, where the bleeding was but slight, this ceased with the simultaneous application of an ice bag to the gastric region and the swallowing of ice pellets. But who can say of such hemorrhages whether they cease spontaneously or under the influence of a drug!

Perforative peritonitis necessitates opium in large doses in the form of a suppository or as an enema, and the use of cold in the form of ice-cold compresses about the abdomen. If we suspect that the stomach is full, we must first try to empty it by the tube after either giving the patient a large dose of morphin or by applying cocain locally. But here it is necessary, under all circumstances, to keep the patient from retching, which may perhaps even enlarge the perforative opening. By this treatment it has sometimes been possible to limit the peritonitis locally, and to bring about adhesions. Operative measures have lately been advised for such cases, and several successful laparotomies have been reported (see below).

Surgical Measures.—For the florid ulcer, as well as for those with recent cicatrices, *surgical aid* has been invoked. Among the first to

favor this were Rossoni in Rome, Nissen in St. Petersburg, and v. Mikulicz in Breslau. At the Surgical Congress (1897) the last author enumerated the indications as follows: "1. When the life of the patient is directly or indirectly threatened by hemorrhage, perforation, inanition. 2. When continuous treatment produces no, or but a temporary, relief, and the sufferings of the patient make his life miserable."

These indications may be somewhat more accurately defined, and operative measures resorted to under the following circumstances:

1. *In stenoses of the pylorus and hour-glass stomach due to cicatricial contraction,* or when continuous internal treatment is powerless to remove the symptoms. This presupposes the consideration that all internal remedies have been thoroughly and exhaustively employed. At the International Congress at Moscow in 1899 I gave the preference to this method of treatment in the but slightly developed field of gastric surgery rather than to operations for cancer, and ascribed to it the most satisfactory and the most lasting results.

2. In *perigastric adhesions* of the stomach to the surrounding organs. Here those adhesions of the greater curvature and at the pylorus are especially to be considered which by volvulus and torsion of the organs and the neighboring structures produce unbearable pain, all anodynes being ineffectual, while an operation at once removes the difficulties which may have existed for years. It is very surprising to see how delicate these adhesions sometimes are. In a case of ours recently operated upon the thin layers of the greater curvature led to the transverse colon, and could be almost completely detached without hemorrhage. Notwithstanding this, they must have been the cause of pains for months, since the patient had no symptoms after the operation.

3. *In gastric hemorrhage.* While the previously mentioned indications are clear and beyond question, provided the diagnosis is positive, in hematemesis it is extremely difficult to say whether and when operative measures are indicated. For even very threatening and very massive hemorrhages are apparently promptly arrested by proper internal treatment. Thrombosis appears with decreasing cardiac power, and it is astonishing to see how rapidly these exsanguine patients recuperate. v. Leube found uncontrollable hemorrhage to be the cause of death in 1 per cent. of his cases. Personally I have never seen a patient succumb immediately to hemorrhage. When Brinton claims 5 per cent. of fatal cases in ulcer due to hemorrhage, only the subsequent consequences, but not immediate death from hemorrhage, can be meant. This makes the decision as to operation difficult, as the results of surgery are by no means brilliant.

Often it is impossible to find the bleeding vessel at the operation, and there are cases in which this cannot be discovered even at the autopsy. Statistics are always very unreliable, for, according to the nature of the case, the successful operations, but not all of the unsuccessful ones, are

published. Kaupe, the most recent author, collected 16 cases up to 1902 in which operation was performed because of acute life-threatening hemorrhage, and 10 of these were discharged as cured; this gives a mortality of only 37 per cent., which figure decidedly differs from the actual facts. The prospects would, perhaps, be more favorable if it could be proven, and to this surgeons have recently called attention, that gastroenterostomy arrests the hemorrhage, that is, prevents its recurrence, without reaching the actual source of the bleeding. Of course those cases only can be considered in which operation was performed during the hemorrhage, for, under other circumstances, we can never be sure that the hemorrhage did not cease spontaneously. Petersen and Maehol actually state: "We have reached the positive conclusion that gastroenterostomy with Murphy's button might save many a patient with severe gastric hemorrhage who would be lost under conservative treatment as well as by any other method of operation." The decision whether Murphy's button is actually a *conditio sine quâ non* for the successful result of the operation I must leave to the surgeons. But, as this technic has been mentioned in the words quoted, I cannot refrain from raising the question (although from principle I do not interfere in the technic of surgical treatment, and do not usually permit myself an opinion) whether, for example, in the case in question, gastroenterostomy antecolica or gastroenterostomy retrocolica is indicated.

During the last few years, i. e., while Murphy's button was still made use of by our German surgeons, I witnessed many operations with and without it, and I am convinced that its advantage of a more simple, and therefore more rapid, technic does not compensate for its well known disadvantages. We are never sure that the button will not fall back into the stomach, and in pyloric stenosis its fate is indefinite; we do not know whether the narrow opening will functionate sufficiently, whether the button has actually passed or has remained in the intestine (its passage may have been overlooked by the nurses), and, provided everything goes well until then, it may even happen that the button is passed some weeks later accompanied by very alarming symptoms which cause the greatest anxiety to the patients and also to the physician not sufficiently familiar with these conditions (for example, after the patient has left the hospital). All of this is obviated in a simple suture anastomosis which, in skilful hands, consumes but little more time than the use of the Murphy button.

4. A further indication for operative relief, and particularly for gastroenterostomy, is given by *persistent hyperacidity*, especially if combined with dilatation of the stomach and weakness of the expelling musculature; of course, only in those cases in which internal remedies have proven ineffectual after a thorough trial.

This occurs in connection with the *sequels of gastroanastomosis* which may influence the function of the stomach in various ways. We might

suppose, *a priori,* that when anastomosis is produced in the deepest area of the stomach a perfectly regular propulsion of the food would result. Some authors, for example, Rosenheim, go so far as to *assume the formation of a new sphincter.* The latter, however, as Petersen correctly remarks, must first be anatomically proven, and this has never yet been done. The possibility of closure of the anastomosis, which has been observed by Rosenheim, Carle and Fantino, or, more correctly, the fact that either water or air is retained in the stomach, merely proves that occasionally a valve-like closure of the opening occurs, not, however, that a sphincter is formed. On the other hand, the consensus of opinion is, and I have often seen convincing proof of this, that the propulsion of the gastric contents after operation is more rapid than before. Certainly if this were not so the result of the operation would be most unfortunate! Whether this is due to an increase of motility or to a freer outflow can hardly be decided. The absence of engorgement of the duodenum has been regarded as indicating an increased motility, for, according to Hirsch and v. Mering, normally, when the duodenum is full, it exerts an inhibitive reflex upon gastric movement. In this theory the fact has been overlooked that in by far the majority of cases in which gastroenterostomy is performed, stenosis of the pylorus occurs as well as a decrease or even a suspension of the propulsion of the ingesta from the stomach into the duodenum. Whether it is advisable in an open pylorus, as Kelling proposes, to close the passage from the stomach into the duodenum by the artificial production of stenosis, and thus prevent the conditions which have just been pointed out, can only be learned from surgical experience, reports of which are still lacking.

There is no doubt that, in many cases, *improvement in the chemism* of the stomach has been brought about. The acidity value, more accurately the value for the excretion of hydrochloric acid, decreases, perhaps less in consequence of decreased secretion than because the bile which regurgitates into the stomach, possibly also the pancreatic juice, neutralizes the acid gastric contents. But that such a decrease of acidity does not take place in all cases may be adduced from my own experiences, which, unfortunately, are not yet published, as well as from the reports of numerous other authors, Dunin, Oderfeld, Kausch, Rosenheim, Rencki, Petersen and others. Nevertheless, the decrease of acidity is so frequent that the above indication for gastroenterostomy in hyperacidity must be mentioned. The regurgitation of bile and pancreatic juice into the stomach—which, under the present method of performing enterostomy between the loops leading to and away, is now usually prevented—gives rise to but slight, if any, difficulty, and this is only perceptible by a bilious taste. Experiments which Joslin undertook in the year 1897 at my suggestion revealed decreased fat digestion, but this, as Nikolaysen later found, is only immediately after the operation, and then is replaced by normal conditions. The ex-

periments at that time, however, were made upon patients who had not been operated upon; what the results would be after this operation is still to be determined.

5. In case of *perforation,* operation whenever possible should be performed in the first twenty-four hours; the later, the worse the chances. In 1895 Pariser could report only 43 cases.

In the year 1896 Weir and Foote compiled a table of the cases of perforating gastric ulcer which had been operated upon, and this showed 78 cases with 23 recoveries, = 29 per cent. In 43 cases the perforative opening was situated upon the anterior wall of the stomach, 11 times upon the posterior wall, and 6 times on the lesser curvature. Accordingly, in the majority of cases this opening may be found and sutured. In 92 per cent. of the cases the history pointed to the diagnosis of gastric ulcer. The importance of early operation is very evident, for in the cases that had existed less than twelve hours the mortality was 39 per cent., in those of twelve to twenty-four hours 76 per cent., and in those over twenty-four hours 87 per cent.

The number of such cases rapidly increased. In 1900, Mayo Robson published statistics compiled from reports of English and American hospitals, comprising 429 cases with a mortality of 55 per cent.; Brunner, however, declares that some cases were counted twice. In the year 1903, Brunner compiled the statistics of 466 cases upon whom operation for gastric perforation had been performed, which gave a mortality of 50 to 64 per cent.; here also the advantage of early operation was clearly demonstrated, for, among the cases in which operation was performed in the first twelve hours, 75 per cent. recovered! It is well known that the chances of recovery for the patient are much better if the stomach at the time of perforation is empty.

Unfortunately, under some circumstances, we cannot determine whether in the case in question we are dealing with a perforative peritonitis due to ulcer of the stomach or not. I have seen a case of this kind terminate in recovery, yet during its course and even later no positive diagnosis was possible.

Brunner collected reports of 17 cases recovering without operation, as against 466 who were operated upon in the same period of time. This number, 17, has been increased by a recent English report of a few cases (*British Med. Journal,* Feb. 20, 1904, H. Whiteford), which, however, is not quite trustworthy.

6. By some authors the *pure, uncomplicated ulcer* has also been designated as suitable for surgical treatment. I do not agree with this. An uncomplicated ulcer heals readily under internal treatment; if healing does not thus take place the ulcer is not uncomplicated, but is combined with changes which resist the regenerative process. Under such circumstances, the indication for operation has been assumed from the fact that

an ulcer may occasionally undergo malignant degeneration. This is analogous to the proposition to operate upon every appendix in which there are signs of inflammation, because subsequently perforation might result. But in the one instance, as in the other, this is but a remote possibility, and in the case of ulcer the danger is very slight, for the number of cases cured decidedly preponderates over those of uncured ulcer of the stomach. As shown above, this proportion is about 2 : 1, but in reality only a small fractional part of unhealed cases undergo malignant degeneration.

Finally, as a general indication for operative interference, immaterial which of the conditions enumerated comes into question, it must be borne in mind that operation only removes the existing pathologic focus, not the predisposition to future relapses. Therefore, the appearance of a new ulcer, another hemorrhage, fresh adhesions, is by no means impossible after a successful operation, and Brasch has described a case in which fatal hemorrhage occurred immediately after gastroenterostomy had been performed. *Consequently those surgeons are right who proceed to operate only when all the remedies of internal treatment have been exhausted, that is, when the condition of the patient permits no further delay. That cases of acute perforation are exceptions to this is self-evident.* In his opinion, and also in the warning against exaggeration of the indications for operation, as expressed by Sahli before the Twentieth Congress of Internal Medicine in 1902, I concur almost absolutely.

It should never be forgotten that the process of healing after these operations is not always smooth and unbroken. I do not refer to the danger of the so-called vicious circle, i. e., the regurgitation of the gastric contents into the false intestinal loop in gastroenterostomy, because this perhaps is avoided by the simultaneous enterostomy, but I must call attention to the occasional development after operation of peptic intestinal ulcers which are a new source of difficulty and danger to the patient. Neumann has recently reported a very instructive case of this kind, and in my opinion given very valuable advice, which is, that in gastric dilatation with pylorospasm and hyperacidity we should first perform jejunostomy and then allow the stomach to rest. Later, if necessary, and under favorable circumstances, gastroenterostomy may be performed.

Mineral Spring Treatment.—Since remote times the hot Glauber Salt Springs, particularly those of Carlsbad, have enjoyed a well deserved reputation, and there is no doubt that the treatment of ulcer at this resort, when carefully conducted and too large quantities of water at one time are not consumed, is frequently crowned by the best results. When the disturbances on the part of the digestive apparatus are relieved, the patient should endeavor to recuperate and gain strength by a sojourn at Franzensbad, Elster, Rippoldsau, Pyrmont, etc., in the mountains or at the sea, with the proviso that he can secure suitable food, which is best when the family does its own cooking. Since, however, many patients

prefer a mineral spring cure to going to bed at home, and many can only avail themselves of the short period of four to six weeks, Carlsbad for these is always the best place, because the opportunity there for dietetic sins is less than elsewhere. After Carlsbad, Neuenahr, Ems, Franzensbad, Homburg and Vichy are to be recommended.

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GASTRIC AND INTESTINAL CARCINOMATA

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When a case of gastric or intestinal carcinoma presents itself before us to-day, we judge it by very different standards from those employed about twenty years ago. At that period, i. e., in the days of Billroth, Péan and Rydygier, our chief endeavor was to make the *diagnosis,* and at that a diagnosis of general visceral cancer. Whether the cancer was located in the stomach and in what part of the same, whether in the small or large intestine, whether in the omentum or the liver, in the gall-bladder or the pancreas, was of scientific but not of therapeutic interest. In every case the patient was doomed; to him accurate proof that this or that portion of his viscera showed malignant degeneration was of no avail.

In the last two decades our views concerning these affections have undergone a complete change the influence of which no physician, however pessimistic he may be regarding carcinoma in general, can withstand. This change, as is well known, has been brought about by *surgery,* and no genius has yet appeared to teach us how to cure by internal remedies. The knife alone can permanently remove the difficulty, or, although very rarely, bring amelioration which lasts for years.

This advance, which is largely due to the high development of antisepsis and asepsis, but also to an enormous impetus to the technic of abdominal surgery, imposes upon internal medicine new obligations.

The diagnosis must be made early. It must accurately define the seat of the affection, it must—when possible—embrace the consideration whether the malignant focus is circumscribed or already generalized. It must decide whether or not any complications are present which may influence the course of the operation, and, finally, it must determine whether at the given moment the patient's strength will warrant surgical interference.

If it were possible in a case of gastric or intestinal carcinoma to decide all these points with certainty, the results of operative treatment would be decidedly more favorable than they are at present, and this would doubtless influence strongly our decision as to conservative or active treatment. Although, in fact, this goal is still remote, our chief endeavor must be to arrive as near as possible to the previously mentioned postulates. For this purpose not only a comprehensive diagnosis, but also the earliest recognition of the affection from all points of view is necessary.

After these preliminary remarks I shall describe a typical case of gastric carcinoma, the delineation of other less typical forms will follow this, and I shall then describe a case of carcinoma of the large intestine and of the rectum; for cancer of the small intestine, on account of its rarity, a brief description will suffice; in conclusion I shall summarize the treatment of gastrointestinal carcinomata.

ETIOLOGY

In examining the *history* of this patient it will be noted that he is about 50 years of age, and up to the time of his present affection he has usually been well; above all, he has had no gastric affection. Until within the last few months he could eat anything and digest it. His present illness dates from this period.

Let us here pause for a moment, since these data are of great importance as indicating the character of the affection. When a patient, who has reached the age at which cancer is likely to occur, tells us that he has never been ill, while his appearance denotes the existence of a serious malady, we must primarily think of a malignant disease. Not that this report is in itself of vital importance, but it indicates the direction in which we must search for the underlying affection.

We have heard from this patient how his ailment gradually developed to its present extent. We are especially interested in learning what the first symptoms were, and whether these gave any indication of the gravity of the condition.

His report, in brief, is the following: On taking food, he has now and then a sensation of pressure which is not actual pain in the pit of the stomach, occasionally there is eructation of gas but not of food, and with this a gradually increasing loss of appetite. This is about all. We see the justification of Brinton's expression in regard to the onset of gastric carcinoma: "Obscure in its symptoms." Nevertheless, any one who has had much experience in the realm of visceral carcinomata will not escape the conviction that this latent and indefinite onset forms the rule to which there are, however, numerous exceptions of practical importance. Among these belongs the *sudden onset with severe symptoms*: In the midst of apparently normal health the patient begins to vomit, and to the surprise of those about him, even of the physician, this continues and steadily becomes more copious so that in a few days, or at most in a few weeks, the patient emaciates markedly, loses his appetite, and becomes feeble and debilitated; briefly, he shows such extreme emaciation that, provided no severe functional or central affection is present—in this connection I refer particularly to the gastric crises and the periodical vomiting described by v. Leyden—without more ado the suspicion of malignant disease is aroused.

Another mode of onset of practical significance is when gastric car-

cinoma is most surprisingly ushered in by decided and threatening *hematemesis* or *melena*. This naturally leads us to think of a simple ulcer of the stomach or of the duodenum, and if etiologic factors are favorable, perhaps also of cirrhosis of the liver or some other form of disease leading to stasis of the portal vein; but the further course shows that a scarcely avoidable error has been made. In connection with the hemorrhage isolated symptoms occur, or all the symptoms of a malignant course may be required to make the diagnosis of cancer more positive from day to day. Practically we may easily assume that a gastric ulcer is undergoing malignant degeneration, but it has been proven with certainty (by May, Ewald and others) that such a course by no means always permits the conclusion of a preceding ulcer. I shall refer to this again in the description of carcinomatous ulcer.

But such occurrences as have been mentioned are usually exceptional. The typical onset, as we unfortunately know, is usually so slightly characteristic that the last thing the patient and his relatives think of is the development of a malignant, incurable disease. And thus it happens that at first, according to their degree of education, they become their own physicians and employ all sorts of home remedies and occasionally purgatives up to a period when every effort at internal or surgical treatment is hopeless. But with careful and comprehensive professional treatment from the first, all concerned will have an expectant attitude. The wise physician will reckon on the possibility of a beginning malignant affection, but because of the absence of definite symptoms, he will refrain from expressing his fears, and not cause unnecessary fright and distress.

At the onset, I purposely mention the difficulties in the recognition of the disease. It will be seen that the demand of many surgeons for an early diagnosis is scarcely justified, for we cannot make a diagnosis before we have the patient. In the chapter upon treatment we shall also consider the fact that, even with an early diagnosis, rapid surgical intervention is sometimes prevented by the stress of circumstances.

After this brief digression, we return to the further course of the disease as described by our patient. He states that in addition to the slight difficulties at the onset disturbances of more serious nature now and then occurred; there was pressure, also pain in the pit of the stomach which occasionally extended from the left side posteriorly into the back, radiating also to the shoulder-blade. Sometimes the patient mentions a symptom that, so far as known to me, is nowhere recorded—a peculiar grating in the gastric region as of two surfaces rubbing against each other. I have heard this symptom described in the same manner by several patients, so that in enumerating the subjective disturbances it appears to me to be of consequence. Now and then vomiting occurs, at first rarely and in slight amount, in the last few weeks more frequently and more copiously. The vomitus consisted of the ingested food which, in the first stages,

showed no alteration in appearance, but lately it has now and then been somewhat brownish. After vomiting the patient is temporarily relieved. At first he lost some flesh; in the last few weeks the weight has declined rapidly, and hand in hand with this there has been a marked decrease in strength; the appetite is wholly lost.

This is the stage in which patients with carcinoma of the stomach usually present themselves to the physician. Clear as the course of the development appears, and strongly as the individual points favor a malignant disease, we must not forget that other severe but not actually malignant processes may have the same or a very similar course of development. I refer, for example, to the cases, recently so accurately studied, of hypertrophic stenosis of the pylorus, the symptoms of which closely resemble those just described. Cicatricial stenosis of the pylorus also may remain latent for a long time, and only when, by a sudden and occasional cause, the musculature is insufficient, may show symptoms which very closely resemble in severity and extent closure by a malignant process.

Only an accurate clinical analysis of the case will decide whether the very suggestive reports of the patient find their explanation in the *objective examination.* While the patient was undressing it was noted that the extraordinary extent of the disturbance in nutrition was most obvious from the upper to the lower extremity. The musculature was soft and flabby, the subcutaneous connective tissue thin, atrophic and without fat, the skin itself could readily be lifted in folds. The *tongue* was coated at the tip and borders. From the present stand of gastric pathology too much importance cannot be attached to the condition of this organ. Various factors here come into consideration (tobacco and alcohol, their use and abuse, fissures, inflammation of the tonsils and pharynx, greater or less cleanliness of the oral cavity) so that the relation between gastric difficulties and the tongue cannot be accurately determined. In contrast to Leser, I do not attach much importance to the small miliary and submiliary angiomata which this patient also showed, for, in my experience, which coincides with that of Gebele, this is not rare in non-carcinomatous cases, particularly in the aged.

After the patient had removed his clothes he was systematically examined in the usual manner. To avoid taking up unnecessary time I shall state at once that the examination of the heart and lungs revealed normal conditions.

In the examination of the *abdomen* I must promptly emphasize the importance of *inspection of the abdomen,* for this method furnishes a fulness of diagnostic aids which can only be recognized by experience. First, the configuration of the abdomen must be noted, and the deviations in both its halves carefully compared. To the right of the median line a relief-like projection is then noted, a slight prominence, which on deep inspiration descended, and upon expiration rose again to its old position.

I shall revert to this later. But another very remarkable phenomenon was noted! From time to time a second relief was seen in the left hypochondrium, rising suddenly and prominently, almost like a mound, from the level of the abdomen, and after a brief time sinking to its old position of rest.

While inspecting the abdomen carefully, and at the conclusion of this phenomenon, a peculiar gurgling murmur was heard.

What does this phenomenon mean? It will perhaps be said that it denotes peristaltic unrest of the stomach, as was first described in a classical manner by Kussmaul, or, as the French term it, "peristaltism." But if this sign which was found to recur at brief intervals and always to run the same course be more accurately examined, we may convince ourselves that it is not from true peristaltic action. On the contrary, it resembles a tonic contraction which does not develop further but remains localized.

The picture is so fascinating that when the phenomenon is at its height we cannot refrain from grasping the prominence with the hands. We feel a hard, contracted mass which resembles the uterus during a pain. No doubt this is the fundus of the stomach. We designate this process *"gastric rigidity,"* and this is analogous to the nomenclature Nothnagel proposed for similar processes in the intestine, to which we shall refer later.

This sign, to which I have recently again called attention,[1] was observed about fifty years ago by no less a one than Cruveilhier,[2] and I cannot refrain from quoting what he says of this symptom in his great "Traité d'anatomie pathol. générale," as follows:

"La dilatation hypertrophique de l'estomac s'observe dans les cas où l'ampliation de l'estomac se faisant graduellement, la contractilité et la force élastique de l'organe n'ayant pas été vaincues, il y a résistance à la distension de la part de la tunique musculeuse, qui se contracte énergiquement sur les aliments, les boissons et les gaz, qu'il contient. *Cette contraction qui se manifeste d'une manière intermittente est facile à reconnaître pendant la vie par un durcissement douloureux analogue aux légères contractions utérines, qui précèdent les grands douleurs de l'accouchement. Pendant toute la durée de cette contraction la forme de l'estomac se dessine à travers les parois abdominales et chez les sujets amaigris on peut en suivre tous les contours à l'oeil et au doigt. C'est à mon avis un grand moyen de diagnostic.*"

In this description the process of gastric rigidity is so masterfully portrayed that little of importance can be added.

My own experience has taught me that several stages of gastric rigidity may be differentiated: 1. Slight contractions extending only to small areas

[1] *Boas, Deutsche med. Wochenschr.*, 1902, Nr. 10.

[2] *Cruveilhier*, "Traité d'anatomie pathol. générale," 1852, T. II, p. 857.

of the fundus, which are felt as increased tonicity lasting but a few seconds (abortive gastric rigidity); 2. Distinct contractions conveying the sensation of marked increase in tonicity, becoming visible as globular prominences, being contractions of a greater or lesser area of the fundus, and terminating with a distinct pressure murmur which is felt by the patient as a more or less painful contraction; 3. Decidedly marked, visible and palpable contractions in the entire extent of the gastric fundus, which continue for a long time, terminate with a distinct gurgle, and produce active pain.

This gastric rigidity is apparently the precursor of peristaltic unrest. Its relation to the latter is that of tonus to tetanus. Occasionally, particularly when the stomach is overloaded, both may be observed side by side.

Gastric rigidity is in my opinion of very *decided diagnostic importance.* It indicates at a relatively early stage that there is an obstruction at the pylorus. Of what nature this is, whether purely functional (spastic contraction of the pylorus) or organic, whether an ulcer, a neoplasm, a gastrolith, an adhesion of the pylorus to neighboring organs, volvulus, etc., can only be decided by continued objective analysis of the individual case. But the fact alone that the patient is suffering from an obstruction at or near the pylorus is not only of great import in the diagnosis but also in the prognosis, and even in treatment.

For this purpose I wish to add a few remarks of practical significance to the discussion of the symptom of gastric rigidity, which is not sufficiently considered in medical literature. "Gastric rigidity," for obvious reasons, may be best noted when the organ is full and it may then be best palpated; but, even then, it can occasionally only be observed with marked friction of the gastric region, preferably with a cool hand or a hand that has been dipped in ice-water. As gastric rigidity does not always occur even under these conditions the examination must be frequently repeated. A slight degree of gastric rigidity can only be determined by much practice, and I therefore advise that the students practise first with cases in which the rigidity is well developed, so as to determine the increased gastric tonus.

I have purposely dwelt upon this phenomenon at some length, for without a complicated apparatus it at once gives the physician reason to suppose that something is out of order in the motor apparatus of the stomach. We have then a certain foundation upon which to proceed, and we know what further steps must be taken to clear the situation.

We shall now recur to the previously mentioned visible *prominence.* We note that it shows inspiratory and expiratory motion, and we shall proceed to palpate it. *As may be readily determined, we are dealing with a hard tumor of uneven surface and about the size of a small orange.*

Apparently this completes the diagnosis. We have before us a tumor

of the pylorus. But we must investigate still further to ascertain the nature of this tumor. Is it movable or adherent? Can it be fixed upon expiration or not?

The movability of a pyloric tumor (for only these can be decidedly moved manually) is so readily proven that we really need say nothing further in regard to it. But only a very few pyloric tumors are movable in all directions like the head of a child in the uterus; in the majority of cases there are some adhesions, and it is then necessary to determine the degree of movability.

If we are in doubt, it is well to inflate the stomach with carbonic acid or air, when it will be possible to determine the position of the stomach, especially that of the greater curvature. If while inflated by air the tumor is still movable, certain locomotor excursions downward and to the right will occur, occasionally also upward and to the right.

Under this method in the case in question we note that inflation by CO_2 (which we prefer on account of its simplicity and particularly because the patient has not yet become accustomed to the prolonged presence of the stomach-tube) affects the position of the tumor but little. Therefore, it is probably adherent to the surroundings.

At this point we must consider an important difference between the expiratory relation of tumors of the pylorus and tumors of the small curvature. The latter, as was first determined by Minkowski,[1] are characterized by *expiratory immovability*. By this sign tumors of the small curvature may readily be differentiated from tumors belonging to other organs, and on the other hand it is also an important factor in differentiating them from tumors of the pylorus. Yet, tumors of the small curvature may lose their expiratory immovability when they become adherent to neighboring organs, but in this case they either do not ascend with inspiration, or they slightly descend.

Judging from the previously mentioned findings the tumor in this case proves to be a tumor of the pylorus, and since it is hard to the touch and the other symptoms also point to a malignant course, we can hardly be wrong in deciding that we have before us a carcinoma of the pylorus.

Nevertheless, although we feel quite certain of our opinion, we shall complete as far as possible the physical examination of the organ, as well as of the other abdominal organs.

Previously, on inflating with CO_2 and simultaneously ascertaining the movability of the pyloric tumor, we observed the position of the greater curvature. It was noted under these circumstances that it extended above the horizontal umbilical line about a handbreadth. We were then unable to determine the position of the smaller curvature as this is only possible on inflating the stomach with air to the extreme limit. These methods,

[1] *Minkowski, Berliner klin. Wochenschrift*, 1888, Nr. 31.

therefore, do not permit us to ascertain whether the stomach is enlarged or descended, or whether both conditions are present.

If we desire accurate knowledge, we must resort to maximal inflations with air, filling with water, or, eventually, the illumination of the stomach. These tests may perhaps have a scientific value, but are of little practical importance.

It is more serviceable to investigate the organs adjacent to the stomach, especially the liver, the intestines, and the mesentery.

As the metastases of carcinoma are principally found in the liver, we should convince ourselves of an enlargement of this organ, or of the presence of nodules therein, and should also palpate the entire intestines and the mesentery, in so far as they are susceptible to the palpating finger; above all we must not fail to ascertain whether or not free fluid is present in the abdominal cavity.

Finally, we must palpate for a moment the left *supraclavicular region* for the possible presence of a so-called Virchow's gland. The occurrence of such glands, sometimes ranging in size from that of a hazelnut to that of a hen's egg, according to the opinion of most authors (v. Leube, Ewald, Riegel, Rosenheim, Mathieu, Hayem and Lion, Hemmeter, Einhorn, Fleiner) with whom I coincide, belongs to the greatest rarities. In regard to the fact that enlargement of the supraclavicular glands (Tarchetti and others) unquestionably belongs to a late period of carcinomatous development, complete unanimity does not exist even among the few authors who ascribe great importance to this symptom.

That enlargement of the supraclavicular glands is not peculiar to gastric carcinoma alone, but to all intestinal carcinomata, need hardly be mentioned. The condition may throw light upon the cases in which the clinical signs of an intestinal carcinoma are obscure. Such instances have been reported in literature, and every one of experience will remember similar occurrences. Under these circumstances, glandular enlargement of the supraclavicular region may aid in the diagnosis.

The same is true of another variety of glandular enlargement which has been observed for a long time, especially in France and also in Germany, but in the main has received but little consideration: The enlargement of the *peri-* and *para-umbilical glands*. Simultaneously, but even alone, the umbilicus may also show metastasis,[1] so that we then speak of umbilical carcinoma. This complication, which Quenu and Longuet[2] a few years ago made the subject of a comprehensive study, is exceedingly rare. Nevertheless, in the course of years I have seen about half a dozen

[1] Cases of primary carcinoma of the umbilicus have been described in literature; they are, however, exceedingly rare.

[2] *Quenu et Longuet*, "Cancer secondaire de l'ombilic." *Revue de Chirurg.*, 1896, T. 16, p. 1897.

of these metastases. That which has been mentioned as clinically important in enlargement of the supraclavicular glands is, *cæteris paribus,* also true of carcinoma of the umbilicus and its surroundings.

This exhausts the leading points of the physical examination.

They are sufficient, as we have seen in the case in question, not only for the diagnosis but also, so far as this be possible, to decide whether or not complications are present. From an examination of the vomited material and from its nature a conclusion, although not an exhaustive one, may be drawn in regard to the *gastric functions,* and it will be apparent that in classical cases of carcinoma of the stomach other methods of examination, particularly an examination of the gastric contents, in a strict sense, are superfluous.

In my opinion these are only necessary when other methods yield insufficient or contradictory results.

As this is often the case, the examination of the gastric contents will be included with the physical examination of the patient in question, although not for the purpose of a strict indication, but for didactic reasons. The patient was prepared for this by withholding food from about eight o'clock of the preceding evening to the following morning.

This method of procedure, i. e., the examination of the empty stomach, is very valuable in daily practice, as by this means without further preparation not only the secretory, but, above all, the motor activity of the stomach may be investigated. In the majority of cases, if the stomach proves to be empty, we may at once administer a trial breakfast and an hour after its introduction the gastric contents may be obtained.

In the case in question the trial breakfast is unnecessary, as the patient's stomach, even early in the morning, contains about one-half liter of residue.

We must first examine the residue carefully.

It consists of a grayish, thin fluid, a mucus-containing mass, as may be noted by pouring the material from one vessel into another, and has an acid but not disagreeable odor. If a portion of the fluid be poured into a test-tube there is no obvious change except that the solid constituents fall to the bottom, but true gas formation which occurs so often in other cases of stagnated gastric contents is not here visible. If the attempt be made to observe gas formation in a ferment tube, no actual production of gas is noted.

Occasionally in the gastric contents or in the vomited material one or several particles of the tumor may be admixed, and if microscopic examination reveals characteristic signs of an atypical neoplasm the diagnosis is virtually certain. It need hardly be stated that macroscopic examination alone under no circumstances suffices, since particles resembling tumor may upon accurate investigation prove to be fragments of mucous membrane, coagulated blood, etc.

CHEMICAL AND MICROSCOPICAL INVESTIGATION OF THE GASTRIC CONTENTS

With this external investigation we now include the chemical and microscopical examination of the gastric contents.

As may be noted from the color reaction (tropaeolin paper, Congo paper) as well as from the results of the phlorogluein, vanillin and resorcin tests, *free hydrochloric acid is not present in the gastric contents.* It was, however, found that litmus paper was decidedly reddened. The gastric contents must contain either much combined hydrochloric acid or acid salts or organic acids, or all of these combined are found. Of these we are chiefly interested in *lactic acid,* especially as its presence may be most easily determined. In fact, as was demonstrated by the iron chlorid test, lactic acid was present in large amounts. Besides lactic acid, other volatile fatty acids are probably present, but their exact determination is difficult and they are of little practical importance.

On the other hand, further to determine the secretory insufficiency of the stomach, the proof that *enzymes,* that is, proenzymes of the gastric contents, pepsin and pepsinogen, as well as lab-ferment and lab-zymogen, are absent is necessary. As was shown by the tests which were made, the previously mentioned enzymes are absent from the gastric contents of the patient in question.

We conclude the chemical examination by testing a portion of the gastric contents for blood, using the guaiac test modified by Weber.

Test for Blood.—As this test has been but little employed in practice I shall at this point briefly describe it. About 10 c.c. of unfiltered gastric contents are mixed in a test-tube with about one-third as much of glacial acetic acid, and the mixture is repeatedly shaken. We then add to it the same quantity of sulphuric ether, after which we shake it, and subsequently note whether the mixture shows any change in color. If much hemoglobin be present in the gastric contents the ether soon changes to a yellowish-brown color somewhat like diluted Tokay wine.

This yellowish-brown discoloration strongly indicates the presence of hematin. The ether is carefully decanted, and ten drops of guaiac tincture *freshly prepared each time* and thirty drops of old, strong oil of turpentine are added. If hematin is present, upon long standing at first a grayish and later a decidedly marked violet color develops in the mixture.

We add to the mixture a few cubic centimeters of distilled water and from ten to twenty drops of *chloroform.* This rapidly takes up the blue coloring matter, and the reaction soon becomes markedly distinct. From the intensity of the blue or violet discoloration we may draw conclusions as to the amount of dissolved hematin present in the gastric contents.

The test made in the case of our patient revealed an extraordinarily profuse amount of hematin in the gastric contents.

Finally, a *microscopic* examination of the gastric contents should be undertaken. Under the microscope numerous immotile rods, partly isolated, partly in clumps or forming angles, were observed; these are *lactic acid bacilli,* which were first described by me, later by Oppler, and accurately studied by Kaufmann and Schlesinger. Furthermore, numerous transverse, striped muscular fibers were noted of which a part were still well retained, now and then starch bodies, also fatty acid crystals and fatty acid needles. Occasionally yeast colonies, isolated or beginning to sprout, are noted, but, on the other hand, the sarcinæ so frequently present in these conditions were absent.

Pus in the gastric contents is a significant finding, and its presence is often revealed by the odor. If the gastric contents are spread out thin upon a black pus basin and carefully examined, as a rule particles of pus will be discovered which may be verified by microscopic examination.

In the preceding I have described the most important macroscopical, chemical and microscopical findings in the gastric contents of the case under discussion. Let us now consider the collective importance of the individual findings.

The least weight is to be attached to the macroscopic investigation; it is true the coffee-ground appearance may strengthen the suspicion of carcinoma, yet it is not absolutely proven. But the previously mentioned particles of tumor tissue unquestionably are in favor of a malignant neoplasm.

More valuable is the chemical finding, and here primarily the absence of free hydrochlorie acid and enzymes, the fact of abnormal lactic acid fermentation, and the presence of blood and pus in the gastric contents are significant.

The absence of free hydrochloric acid (which, as is well known, was discovered in Kussmaul's Clinic in the year 1879 by van den Velden), even to-day is looked upon as a relatively certain and decisive sign. It is of very frequent occurrence in gastric carcinoma; it was present in 77.5 per cent. of my cases, and other observers give even higher figures. But gastric carcinoma is not the only affection which shows the absence of hydrochlorie acid. This symptom is observed in maladies of an entirely benign character: In chronic gastritis, in amyloid degeneration, in severe forms of anemia, in poisoning by sulphuric and hydrochloric acids, in so-called achylia gastrica, in hypertrophic pyloric stenosis, and in rare cases of ulcer of the stomach; moreover, deficient hydrochloric acid is found in carcinoma of other organs, for example, of the œsophagus, of the uterus, of the breast, and of the rectum.

Inversely, free hydrochloric acid is occasionally found even in conspicuous excess in well developed cases of gastric carcinoma. An excess of hydrochlorie acid is especially frequent in carcinomatous ulcer, to which we shall refer later. But also in undoubted gastric carcinoma, particu-

larly of the pylorus and at the onset of the disease, we quite often find hydrochloric acid.

Of greater decisive importance is the occurrence of large amounts of *lactic acid.* Arthur Schiff[1] compiled the figures of Hammerschlag, Strauss, Schlesinger, Kaufmann and Ekehorn, which show that 84.4 per cent. of *gastric affections running their course with lactic acid fermentation are gastric carcinomata.* According to the same compilation, the investigations of Boas, Hammerschlag, Rosenheim, Lindner, Kuttner, Robin, Strauss, Kaufmann and Schlesinger show that lactic acid occurs in 73 per cent. of all gastric carcinomata. Higher figures were given by Croner,[2] that is, 78.5 per cent., and Rütimeyer,[3] 75 to 80 per cent.

On the other hand, it cannot be denied that lactic acid fermentation in the stomach occurs in quite a number of non-malignant gastric affections; for example, in hypertrophic pyloric stenosis, gastritis gravis (Rosenheim), in pernicious anemia with gastric atrophy (Ewald), and in other rare cases which, however, do not clinically present the picture of gastric carcinoma.

As the presence of lactic acid occurs only with stasis of the gastric contents and the absence of hydrochloric acid, it is obvious that deficiency in lactic acid by no means excludes gastric carcinoma.

The positive finding only is of importance, not the negative.

Besides the chemical changes previously mentioned the absence of *pepsin and lab-ferment* is to be emphasized, to which Hammerschlag, Ewald, Kuttner and others attach great value. This, however, always occurs when the presence of lactic acid is combined with the absence of hydrochloric acid, so that proof of the absence of enzymes at most only confirms the previously mentioned anomalies or complements them.

Occult Gastric Hemorrhage.—In my experience some weight must be attached to the evidence of *occult gastric hemorrhage.* This may be detected in the gastric contents or, if for any reason not thus perceptible, may be demonstrated in the feces by means of the previously mentioned method (page 230).

Since Ewald and Kuttner called attention to the occurrence of these slight hemorrhages, I have recently had an opportunity to study this subject carefully, and in numerous cases of gastric carcinoma have determined that in these diseases occult hemorrhages are exceedingly common. Thus, associated with my assistant, Dr. Kochmann, in 67 cases of gastric carcinoma we found 65 hemorrhages, and in the overwhelming majority of cases the bleeding was occult.

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[1] *Arthur Schiff*, "Die Diagnose des Magencarcinoms," "Sammelreferat nach den Arbeiten der Jahren 1894–1898." *Centralbl. f. die Grenzgebiete der Medicin u. Chirurgie*, 1898, Nr. 12 and 13.

[2] *Croner, Mittheilungen aus den Grenzgebieten*, 1899. Bd. V, p. 405.

[3] *Rütimeyer, Correspondenzbl. f. Schweizer Aerzte*, 1900, Nr. 21.

However, this finding is not pathognomonic. We have noted the condition in a large series of observations, most numerously in cases of benign dilatation, also in hypertrophic pyloric stenosis and in ulcer of the stomach, but not, however, in positive cases of chronic gastritis and achylia. Naturally, even in these cases, fresh visible hemorrhages may appear as capillary hemorrhages which, however, as a rule, are of artificial origin. Continuous hemorrhage in chronic achylia or gastritis without conditions of stasis belongs to the greatest exceptions; in carcinoma, however, it forms the rule.

The differentiation of a benign from a malignant pyloric stenosis by demonstrating the presence of blood is not readily made. But we may state that under these conditions the reaction in carcinoma is decidedly more marked (saturated blue) than in benign processes (delicate violet).

Microscopic examination has a certain importance inasmuch as it reveals the thread bacilli of lactic acid fermentation, and thus confirms the presence of the latter. *Sarcinæ*, in my experience, are of no diagnostic significance in gastric carcinoma. They are absent, as a rule, but not invariably, in marked lactic acid fermentation, and, *vice versa*, with the finding of free hydrochloric acid and marked stagnation they are almost always present in carcinoma.

Pus in the gastric contents is by no means a rare symptom; usually it is found admixed with blood. Although pus may occur in the stomach from various causes (gastritis phlegmonosa, abscesses) and is especially prone to originate from the upper air passages, the continuous finding of amounts of pus, particularly if the conditions mentioned may be excluded, is a valuable sign of carcinoma.

Besides the gastric contents, the composition of the blood in gastric carcinoma has received considerable attention; in the main, however, the results are unsatisfactory. We often find, but not constantly, a marked diminution of hemoglobin and of the erythrocytes. The opinion promulgated a few years ago by Schneyer [1] that there is an *absence of digestive leukocytosis* in gastric carcinoma was found to be incorrect after further examination (by Hassmann, Hoffmann, Sayler and Taylor, Chadbourne, Dolmatow, Marchetti, Rencki and others).

From this description it follows that absolute proof of the presence of a gastric carcinoma, aside, perhaps, from the very rare finding of particles of the tumor which is only possible in well advanced cases, cannot be obtained from the investigation of the gastric contents. Only most careful consideration of all the clinical signs, among them the physical, particularly the tumor, can protect us from an erroneous diagnosis.

With this we reach the important and difficult chapter of the diagnosis of gastric carcinoma.

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[1] *Schneyer, Internationale klin. Rundschau*, 1894.

GASTRIC CARCINOMA

DIAGNOSIS

So long as we are dealing with cases similar to the one just described, the diagnosis of gastric carcinoma is one of the easiest in the realm of internal medicine. Difficulties beset us only when the onset is indistinct and the course atypical, when the symptoms differ in important points from those just described, when important objective factors are either absent or indistinctly developed.

We may differentiate two categories of atypical development: First, those in which a tumor in the stomach is not palpable at the moment of examination; secondly, if the tumor be present, it does not have the characteristic properties of a gastric tumor, and particularly of a malignant gastric tumor.

We shall now concern ourselves with the first class of cases. Daily observation gives us many examples of this kind.

We see, for instance, a patient who for a short time has complained of dyspeptic symptoms: Gastric oppression, anorexia, now and then drawing pains in the epigastrium or in the fundus, occasionally also in the back, eructations, emaciation, a feeling of diminished dynamic activity, etc. Palpation may perhaps reveal slight sensitiveness to pressure, but other than this no objective signs are observed.

The suspicion of a pernicious disease is aroused, but we may also be dealing with a severe form of chronic gastritis or even with one of those not infrequent forms of nervous dyspepsia which in the course of time lead to an extreme degree of marasmus, and thus very closely simulate the picture of carcinomatous cachexia.

In another series of carcinomata we observe an entirely different course. The disease begins suddenly as a severe gastritis, or like an acute gastric dilatation: In the midst of health the patient begins to vomit, or, after a more or less obvious cause, once or several times large or small amounts of ingesta are ejected; but notwithstanding careful regulation of the diet and the use of all remedies calculated to ameliorate the condition, the symptoms do not yield but even increase. The patient permanently loses his appetite, cannot take even the most carefully prepared food, and in a few weeks shows a threatening state of inanition.

Or after indistinct prodromes the patient is suddenly attacked with profuse hematemesis or melena. We first think of a latent ulcer and institute appropriate treatment; the hemorrhage ceases but the patient does not recover his health, he rejects his food, repeatedly vomits, sometimes food but occasionally also fresh blood, and the case begins to assume a serious aspect.

In other cases, the patient shows indistinct dyspeptic symptoms with fever which is not characteristic, nor does it conform to any of the well-

known types. We think of cryptogenetie pyemia or an irregular attack of enteric fever, of malaria, of tuberculous peritonitis, etc., and finally section shows the presence of a gastric or intestinal carcinoma which was insusceptible to palpation.

This shows but a few forms under which carcinoma may be masked. If we undertook to enumerate them all we should lose ourselves in a chaos of conditions which could not be examined.

Somewhat less intricate but still obscure is the second category of cases in which a tumor is present but in which doubts arise as to whether it belongs to the stomach, or in which the history or malignancy is uncertain. We mention, for example, the difficult differentiation of an old ulcer with thickened borders from a carcinoma or a carcinomatous ulcer, furthermore, the recognition of a callous, indurated perigastritis, a hypertrophic thickening of the pylorus, a hard gall-bladder filled with stones, a gastrolith, a lipoma, a papilloma or myoma, not to speak of sarcoma of the stomach.

In these and numerous other cases which cannot be mentioned here all methods of diagnosis, particularly the examination of the gastric contents, must be utilized, and, as I must admit, frequently without success, to clear up these doubtful conditions. But, nevertheless, cases remain in which nothing but the further course of the disease, and often not even this, but only laparotomy or the autopsy, will decide the question.

To proceed, we must mention some other methods which now and then are employed in complementing the diagnosis.

First among these is the examination of the patient under *anesthesia.* By this means doubtful tumors whose presence in the various abdominal organs cannot be determined with certainty may be better recognized or localized. However, in consequence of more thorough technic in palpation, examination under an anesthetic has in the last few years fallen into disuse. It is best reserved for the cases in which active tension of the abdominal walls from marked meteorism, etc., makes it impossible to obtain a clear idea of the condition of the viscera.

The second method is *gastroscopy* which, however, up to the present time has secured no permanent place in the diagnosis of gastric pathology. Invented by v. Mikulicz, and further developed by Rosenheim, Kuttner and Rewidzoff, this method has been chiefly employed in the last few years by G. Kelling. Kelling is of the opinion that gastroscopy enables us to make the diagnosis in doubtful cases of gastric carcinoma, and in other instances to make it early (see below). We must wait and see what the technic and possibilities of gastroscopy achieve in the future, before we can look upon this method as positive and decisive.

So-called *exploratory laparotomy* has also been employed as a diagnostic aid. As a matter of fact every laparotomy is an exploratory incision, as only this gives us accurate knowledge as to the seat of the tumor, its

physical composition, its relation to neighboring organs, the existence of complications, etc.

In this respect, exploratory incision is in truth the best and most certain diagnostic method. But this opinion is not generally held nor the fact recognized, above all among the laity. Although with the great development in technic as well as in antisepsis and asepsis the risk is not great, nevertheless the method is rejected even at the cost of a positive diagnosis. In these instances laparotomy is frequently begun as a therapeutic measure and ended as an exploratory operation.

I believe an exploratory incision to be unjustifiable *when the diagnosis is wholly obscure or when we entertain the suspicion of a malignant process but cannot possibly determine the organ affected.* Here from a simple exploratory incision a lengthy, severe, and occasionally ineffectual operation may result. In describing the indications for the operative treatment of gastric carcinoma we shall again revert to this subject.

From what has just been stated the impression will be given that in the majority of cases of gastric carcinoma a clear diagnosis can be made. Much more perplexing is it to determine at the onset with certainty or probability the exact seat, the extent of the process, the presence of metastasis, a beginning ascites or other complications. In so far as we are dealing with objectively demonstrable changes a careful examination will naturally assist us. Other very frequent occurrences, such as metastasis of the liver, or of the mesentery, etc., sometimes escape recognition. *A diagnosis entirely comprehensive in this respect is impossible.*

Intimately related to these difficulties is the much discussed question of the *early diagnosis of gastric carcinoma.* Even the prior question, "What do we understand by early diagnosis?" is not easily answered. It has been said "that a diagnosis is made early if no palpable tumor is present." This is generally true. But the facts show that even in Billroth's time radical operation was attempted for gastric carcinoma, which proves that this requirement was not absolutely a prior condition for successful surgical treatment but also, inversely, what every one of experience knows, that the impossibility of feeling a tumor is no proof of conditions favorable to operation. Neither will the time which has elapsed since the onset of the disease furnish us any clue to the distribution of the process, as groups of tumors which grow rapidly and also such as grow very slowly have been observed.

The chemical findings, particularly the triad, stagnation, absence of hydrochloric acid, and presence of lactic acid, will in some of the individual cases enable us to make an early diagnosis even although the tumor elude discovery, although isolatedly there are benign processes with the same chemical finding, and, notwithstanding the fact that the presence of this triad of symptoms indicates conditions already very unfavorable for the removal of the tumor.

Nevertheless, in the points above mentioned, collectively at least, and so long as no better criteria exist, these factors in a large number of cases permit an early diagnosis.

The usually very decided period of latency forms the greatest obstacle in the discrimination of gastric carcinoma. In this stage the patients have but relatively few symptoms, do not consult a physician, and resort to home remedies. Certainly most of the carcinomata which we meet are already well advanced and manifest conspicuous symptoms, above all, a palpable tumor. In this condition gastroscopy—even should the technic become more simple—will not materially aid us. Nothing is easier than the diagnosis of rectal, uterine or mammary carcinomata, and yet in these we hear of precisely the same perplexities on the part of the surgeons and gynecologists as in the case of gastric carcinoma.

Nevertheless it would be a great mistake to allow these obstacles to daunt us, and to relinquish our endeavors to make an early diagnosis. The marked successes reported in literature resulting from early operation after an early diagnosis of gastric carcinoma should encourage us, even with the basis of our present methods, always to attempt to diagnosticate the disease as soon as possible.

A special form of carcinoma still to be described is *carcinomatous ulcer*. Previously investigated by Rokitansky and Dittrich, carcinomatous ulcer has recently been thoroughly studied by Hauser. Opinions as to the frequency of the transformation of a simple ulcer into carcinoma are to-day widely asunder. According to Lebert, 9 per cent. of all gastric cancers are due to this cause. Rosenheim found it only in 5 to 6 per cent., and Ewald has lately declared carcinomatous ulcer to be far more frequent than has up to this time been assumed.

According to my experience a positive diagnosis of carcinomatous ulcer *intra vitam* is impossible. The chemical examination of the gastric contents utterly fails us. The excess of hydrochloric acid which Rosenheim previously believed to be decisive is found, as we now know, also in carcinoma without ulcer. I have repeatedly observed in carcinomatous ulcer severe, rapidly succeeding gastric hemorrhages. Yet even these are not decisive. We should always think of *carcinoma ex ulcere* if positive symptoms of ulcer have for a long time preceded the process.

COURSE

The course of gastric carcinoma depends upon various factors, primarily upon the nature and seat of the tumor. Experience has shown that there is a gliding scale for the malignancy of carcinoma; that, for example, adenocarcinomata grow more slowly and less frequently develop metastasis than the rapidly growing, speedily decomposing medullary carcinomata which readily promote the carrying of carcinomatous products.

Scirrhus is again characterized by its tendency to advance rapidly and cause stenosis, colloid carcinoma by its unlimited growth.

Possibly *the seat* of gastric carcinoma is of even greater significance in the course. It was formerly assumed that the point of predilection was the pyloric region. More recent experience, as well as operations and autopsies, shows this to be no longer true. Carcinomata of the smaller curvature are not less frequent than those of the pylorus, and it is even likely that, similar to the course in ulcer, the smaller curvature is the preferred seat for carcinoma.

No matter how this may be, the fact is generally accepted that the nearer the carcinoma to the pylorus the more rapid and more serious the course of the disease. Under these circumstances the malady may set in with elementary force and in an exceedingly short time, even a few weeks, cause death (*acute carcinoma*). In opposition to this are the slowly growing carcinomata with numerous variations which are clinically ill-defined, and, according to whether improvement or aggravation appear, keep the patient and the physician wavering between hope and fear.

The cause of this latency is easily recognized if we remember that in carcinomata not situated in the pylorus vomiting is absent or only present to a slight extent, that hemorrhages also are but moderate, and that, finally, the ingestion of food and its assimilation are far less damaged than in the first mentioned group.

Under such circumstances the patients may for a time gain in weight and subjective activity; nevertheless, the outcome shows that the physician was correct in his grave prognosis.

It need scarcely be mentioned that the course of the pathologic process is influenced to a decided extent by the most varied complications, such as ascites, proliferation into the peritoneum, external rupture or rupture into the intestine, metastatic pleurisy, etc.

We see from this that in a given case the determination of the duration of the course is beset with great difficulty. However, it may be estimated on the average at a year to a year and a half. If this period has elapsed we will rarely go astray in excluding a malignant process.

COMPLICATIONS AND SEQUELS

The complications which may occur in the course of gastric carcinoma are numerous. Some of these are due principally to the nature of the affection, and for this reason are not of great clinical interest. Others, however, require a brief description.

I must first call attention to the *fever* which occurs in carcinoma; this was mentioned by Wunderlich in his well-known text-book, and has been more accurately described by Hampeln (1884).

Carcinomatous Fever.—According to Freudweiler, who made a thor-

ough study of carcinomatous fever in 475 cases of carcinoma in the Zurich Clinic, this symptom was present in 117 cases (i. e., in 24.6 per cent.), and was not due to complications; rarely (that is, in 1.5 per cent. of all cases) the fever was almost continuous, usually it was remittent or intermittent (in 10 per cent. of the total number of cases); much more rarely it was typically intermittent and resembled malaria (in 3.6 per cent. of all cases, in 14.5 per cent. of febrile carcinoma); in the latter case, the fever was not so typical as in malaria, often it was of an inverse type, the time of its appearance varying on different days as did the duration of the febrile period; in a number of cases the temperature rose but gradually instead of suddenly as in malaria. With relative frequency (in 38.5 per cent. of the fever cases and in 9.3 per cent. of all cases of carcinoma) there were isolated rises in temperature which did not last longer than three days.

In the majority of the patients the condition was that of ulcerative carcinoma so that the fever was attributed to secondary infection; in the minority, however, no ulceration could be detected at the autopsy. Therefore, carcinoma in itself, similar to tuberculosis and lympho-sarcoma, has pyrogenetic properties.

Inversely, as Freudweiler has shown, carcinoma may also run its course with subnormal temperature. In 47.8 per cent. of his cases temperatures below 96.8° F. were repeatedly found.

Coma Carcinomatosum.—A second important complication is *coma carcinomatosum* which was first described by v. Jaksch, then by Senator, Riess, G. Klemperer and others. *Acetone* was first found by v. Jakseh in the distillate from the urine, and Klemperer later detected *oxybutyric acid.* Coma carcinomatosum resembles diabetic coma except that, in my experience, its course is not so rapid. In one of my patients with rectal carcinoma, in whom a few months previously colostomy had been performed, the comatose condition lasted very nearly two months. Whether in these cases, as is exceedingly likely in diabetes, there is an acid intoxication, or whether specific toxins generated by the carcinomatous poison are operative, is not yet known.

Tetany is very much more rare than the two complications just mentioned. This form of tetany does not differ from that occurring in benign pylorie stenosis.

In conclusion we must mention the dropsical symptoms in carcinoma (edema and ascites), venous thrombosis and metastases in the liver which, according to Lebert, occur in 50 per cent. of all the cases, also metastases of the pleura and of the lungs, and finally the appearance of multiple neuritis.

PROGNOSIS

The prognosis, as I need hardly mention, is very unfavorable provided surgical intervention does not soon interrupt the course for a time (un-

fortunately, however, not for a very long time). As we shall show in the chapter upon therapy, internal treatment may bring about amelioration of many of the symptoms and increase the strength, but the unfavorable outcome cannot be averted. When no complications are present, the prognosis depends mainly upon the nature and seat of the tumor. As I have already mentioned, carcinomata which do not implicate the pylorus run a much slower course, I might almost say a more benign course, than carcinomata which cause stenosis. As regards danger to life, such carcinomata may be prognostically designated as more favorable, but with the limitation that under a radical operation the chances of success are even less than in those of the pylorus.

CARCINOMA OF THE INTESTINES

Proceeding downward from the stomach we meet with carcinomata everywhere but—and this is of great importance—by no means with the same frequency. While carcinomata of the small intestines are extremely rare, carcinomata of the rectum are about as numerous as those of the esophagus. Between these, carcinomata of the large intestine are found; and here also the curvatures are the points of predilection. From this it is evident that, a fact to which Virchow called our attention, those portions of the digestive canal through which the contents move but slowly and sluggishly are particularly predisposed to the development of malignant tumors.

SYMPTOMS

I shall first describe a typical case of carcinoma of the large intestine, then one of the rectum, and in connection with these shall briefly delineate the symptomatology of carcinoma of the small intestine.

The patient was a man in the forties and, except for the diseases of childhood and other mild affections, he had up to the present time always been well. In particular he had never complained of gastrointestinal disturbance. Here we observe the abrupt onset in the midst of perfect health to which I called attention when describing carcinoma of the stomach.

The patient stated that the first symptom was stubborn constipation, so that at the onset of the affection he was compelled to resort to active purgatives to regulate the function of his bowels. Following the constipation attacks of severe colic soon appeared, first at long intervals, and later every two or three days. Finally, he complained of marked loss of weight, lassitude and inability to follow his occupation.

It is evident here that the onset of the affection was but slightly characteristic.

At first a mild coprostasis, a flatulent colic appears, such as we are

accustomed to see every day. But, under these circumstances, it is well not to be too optimistic, even in cases in which there is no serious indication of a malignant affection, particularly when an obvious loss of weight cannot be attributed to alimentary processes.

In every case it is well to bear the patient in mind. Repeated weighings should be made, and the physician should not be content until he has decisive evidence whether a mild, or, *vice versa,* a very serious malady is present. Too great pessimism will do no harm, especially if it should later give way to positive optimism.

So much as to the onset of the disease. Let us now discuss its further development. In the last few weeks the intestinal cramp increased to an unbearable degree. The patient was compelled to take narcotics. Purgatives which at first had been active were now ineffectual even in large doses. Stronger drugs were all the time taken, and finally drastics were employed, without relief. The feces no longer appeared in solid, cylindrical masses as formerly, but in the shape of small scybala, frequently admixed with mucus or as a small quantity of thin fluid, or, finally, in this or that form. The patient has not noticed blood in the dejecta. Upon being questioned he reports that he frequently noted simultaneously with the attacks of pain, but often also without these, a distinct, intestinal rumbling similar to the dripping of a fluid, and that this was even audible at some distance. Very observant patients will occasionally state, upon being asked, that occasionally, especially if active pains are present, the prominence of some portions of the intestines has been perceived.

Although these symptoms are by no means positive signs of the nature of the affection, nevertheless it is wise not to place too low an estimate upon them. I can positively state that, from a history of this kind all the details of which have been taken into consideration, I have made a diagnosis of carcinoma of the large intestine even before the patient undressed himself for a physical examination.

Absolute certainty, above all, is furnished by the clinical findings. I shall not allude to the general symptoms, which were discussed in the previous chapter on visceral carcinoma, but shall only consider what may be demonstrated locally.

In intestinal carcinoma the finding of a well-characterized *tumor* is unquestionably the most positive and decisive symptom.

Let us discuss the local findings in the patient in question. As was readily observed, there was an apparently hard but slightly movable tumor about the size of a mandarin in the cecal region, and this could be palpated.

Does the tumor belong to the intestine, and to which part? Are we dealing with a malignant tumor? What is its degree of movability? As these questions arise in every case that is not absolutely clear, their immediate investigation is imperative.

The fact that the tumor belongs to the intestinal canal may, as a rule,

be determined without great difficulty from the history in connection with the other clinical findings. Nevertheless, sometimes perplexities arise which greatly annoy the diagnostician. A displaced lobe of the liver, hepatoptosis, with a gall-bladder full of stones or showing carcinomatous degeneration or dropsy, normal but low-lying kidneys, renal tumors, tumors of the omentum, and even tumors of the pylorus may, under some circumstances, simulate neoplasms of the large intestine. In the chapter devoted to diagnosis I shall enter upon these details more minutely.

To prove whether a benign or a malignant process is present is by no means always easy. Chronic perityphlitis with decided induration, tuberculous thickening of the cecum, not to speak of other benign tumors (fibromata, lipomata, myomata, etc.), may resemble malignant neoplasms to such an extent that the true condition is recognized only after prolonged observation, often only at the operation or at the post mortem.

In regard to movability, the law generally obtains that in intestinal tumors this should exist to a high degree. This general rule does not coincide with my experience. I can only admit that the most extreme grades of movability are observed in carcinomata of the large intestine; yet, when a great number of cases are studied, we find only slight, passive locomotion to be present. In the development of the tumor adhesions to neighboring organs very soon form.

For distinct passive movability in inflation of the rectum with gas we have a method which is usually serviceable, but the intestines must not be too greatly distended with gas for, if this be the case, the tumor is covered by the intestines and is palpated with great difficulty.

In the case in question it was readily determined that the tumor was hardly movable, and I may add that upon repeated investigation its position was unchanged. The inflation of the intestines per rectum gave no other results.

Where there is undoubtedly a hard tumor of the large intestine, as well as other points in the clinical history, the clinical picture of carcinoma of the large intestine is sufficiently characteristic; there can be no doubt of the diagnosis. Other factors which we shall later consider at most serve to complement the pathologic picture.

Very frequently, however, the symptoms are complicated. A tumor is absent. There are signs of a malignant intestinal affection but without positive indications as to its nature. Frequently we note a symptom which we also find in the case under consideration: The picture of *chronic intestinal stenosis*. As we have in this volume a masterly description by Nothnagel of chronic intestinal stenosis (which see) I limit myself to a few salient points which relate particularly to carcinoma of the intestine.

Intestinal Rigidity.—In this condition the severe *attacks of obstruction* accompanied by severe colic, occasionally by vomiting, which have already been described are subjectively decisive. Objectively, we must

consider meteorism, suprastenotie sueeussion, and the visible, tetanie, intestinal eontraetions to whieh Nothnagel has given the appropriate name of "*intestinal rigidity.*"

In well-developed eases of intestinal stenosis meteorism is rarely absent, but is present in varying degree aceording to the individual ease. Sometimes it is localized, and permits a somewhat aceurate recognition of the seat of the intestinal stenosis, or it may be general in the stage of intestinal paralysis, that is, of threatening ileus, and may then be indistinet or obliterated.

Intestinal rigidity is the clearest and the most objective symptom of intestinal stenosis. In well-developed eases we note from time to time that the eoils of the intestine above the stenosed area eontraet tetanieally with sensations of aetual pain, they beeome hard and stiff, and remain in this condition for some time. With a distinctly audible murmur that I have designated a pressure murmur, this eontraetion suddenly relaxes, or to a eertain extent remains permanently. These are the elassieal types of intestinal rigidity, but there are numerous deviations. The intestine may eontraet, for example, but not tetanieally, it may become hard and only show an inerease of tonieity similar to what we have observed in the stomach, or it rises slightly in the abdominal wall and rapidly sinks without produeing aeoustie phenomena in its state of relaxation.

The extent of the stiffened intestinal areas varies greatly, according to the seat and the extent of the stenosis. Where stenosis is very marked and provided the seat is in the eeeum or in the aseending eolon, although rarely when deeper, we note how the small intestines rise one above another like the pipes of an organ, eovering the entire abdominal surface, as has been graphieally deseribed by Nothnagel.

It is seareely possible to eonfound this intestinal rigidity with the indistinet peristaltie motion whieh we meet with physiologieally in well-developed diastasis of the reeti, or in motility neurosis of the intestine (tormina ventrie. nervosa, Kussmaul). The absenee of pain, of rigidity, of eonstipation, of intestinal splashing, of a pressure murmur, indicates without further eonsideration the marked differenee.

Although intestinal rigidity points only to the existence of stenosis, and only exceptionally to its seat, we are frequently able to make a diagnosis of intestinal eareinoma as well as from the remaining elinical eourse even in the absence of a tumor. Naturally, this is not always an early diagnosis but, under some eireumstanees, the eonditions may be so favorable as to permit a radical operation which may bring relief to the patient for a long time.

In another group of intestinal carcinomata we find no symptoms of intestinal stenosis; these are the cases which from the onset show no annular band formation with eieatrization, but in which diffuse, infiltrating, ulcerating tumors are found, as in the curvatures of the stomach.

The symptoms in these cases—provided a well-developed localized tumor be absent—vary greatly, according to the stage, the seat, and the extent of the process. It may happen that for weeks and even for months positive intestinal symptoms are entirely lacking. Or, if they are present, they point to a severe intestinal affection the nature of which for a long time cannot be discerned.

In such cases periodic, persistent *investigation of the feces* may in the course of time show the true nature of the condition.

The investigation of the feces in intestinal carcinoma has not the same clinical importance as the examination of the gastric contents in the diagnosis of gastric carcinoma. But a few factors are, nevertheless, significant. Among these is the single or repeated admixture of blood with the feces. This may introduce intestinal carcinoma, or may appear in its course, or may form the terminal symptom.

The hemorrhages may be copious, or may appear as small specks which are hardly visible, or, finally, they may be occult and only detected by chemical examination (guaiac test) or by the spectroscope. Kochmann and I observed such occult hemorrhages in two cases of carcinoma of the colon, and greater attention will, therefore, subsequently be devoted to this symptom.

In the main, however, copious intestinal hemorrhages are not common symptoms of intestinal carcinoma. Such an experienced investigator as Treves [1] calculates their frequency at only 15 per cent. Whether, however, smaller hemorrhages which readily escape recognition by the patient and the physician are not more frequent is yet to be determined.

Besides pure hemorrhages, admixtures of *blood and pus* have been observed by other authors as well as by myself. I have repeatedly noticed these in deep-seated intestinal carcinomata (in the sigmoid flexure) which offer more favorable conditions for the retention of pus corpuscles than, for instance, the cecum or even the small intestine.

That mucus also is sometimes admixed with the feces is readily conceivable if we remember that the carcinomatous neoplasm produces in a large portion of the intestinal tube a condition of catarrhal inflammation. In a case of carcinoma of the ascending colon near the cecum, determined at the autopsy, I observed even a well-developed membranous colitis.

In regard to the form of the feces, it has previously been briefly stated that various deviations from the normal may be observed.

Thin, flattened, tape-like feces have been designated as typical of stenosis. We know to-day that this is untrue, not because normal feces may temporarily appear in stenosis nor because the changes mentioned indicate the formation of stenosis. Only one condition of the feces is never found

[1] *Treves*, "Intestinal Obstruction," London, 1899.

in carcinoma causing stenosis, namely, a *persistently well-formed, cylindrical shape.*

In very isolated cases of intestinal carcinoma, just as in gastric carcinoma, shreds of the tumor are found in the feces. The few observations at hand do not permit a conclusion as to the clinical value of this symptom; nevertheless, this much is certain, that sequestration only occurs in well advanced cases.

In this description I have considered mainly the high-seated carcinomata of the large intestine. The deeply-seated ones, therefore those from the flexura colico-lienalis downward, are marked by certain peculiarities which I must briefly indicate.

Here *tenesmus,* which we must consider in detail under the description of carcinomata of the rectum, becomes prominent, not only tenesmus recti alone but combined with tenesmus vesicæ.

Rectal Carcinomata.—Occasionally the latter may be so prominent that we are inclined to think of an affection of the bladder. If a tumor be present it is situated either in the region of the sigmoid flexure or closely adjacent to the bladder where it may be felt below or beside this organ. In one of my cases the tumor was situated at the right side of the bladder, and was therefore thought to be a cecal tumor. Occasionally, however, carcinomata of the sigmoid flexure, as Körte correctly remarks, are not susceptible to palpation, that is, when their situation is intermediate, or just above the rectum. They cannot be reached by digital examination either from above or below, and offer great difficulty in diagnosis as well as in surgical operations. Carcinomata of the sigmoid flexure lead immediately to the description of the most deep-seated intestinal carcinomata, those of the *rectum.* We shall proceed to consider these by quoting a concrete case.

The patient, as was at once apparent, showed no cachexia, nevertheless the history denoted that we were dealing with a serious affection. He was 52 years of age. He stated that his mother perished after several operations for a tumor of the uterus, his father died from apoplexy. The patient gave an excellent history of himself up to the time of the present disease, which began about five months ago with a slight sensation of pressure and inability to evacuate the bowels naturally. The feces were very frequently admixed with mucus, gradually tenesmus became more marked, and every three or four hours the patient was compelled to seek the toilet, frequently without other result than thin fluid feces admixed with mucus, and even then showing traces of blood.

Thinking he had intestinal catarrh he resorted to a diet consisting of non-irritating gruels and soups with the result that tenesmus was produced and his strength and weight decidedly decreased.

Among the symptoms tenesmus became prominent and this at once indicated that the seat of the affection was in the rectum. Of course in all

cases in which this symptom is mentioned we do not invariably at once think of carcinoma, as the same is complained of by patients with hemorrhoids, with acute and chronic proctitis, with ulceration in the rectum, with prolapse of the rectum, with so-called fecal impaction, and even in purely nervous spasm (proctospasm) this symptom is by no means rare. *Nevertheless, whenever we have the symptom of tenesmus a careful digital or ocular exploration of the rectum should at once be made.*

Before describing the patient's condition we shall first determine whether physical examination of the thoracic organs and external palpation revealed any anomaly. This was not the case except for one suspicious point. We found that the liver was decidedly enlarged. It projected beyond the border of the ribs about three fingerbreadths, and the border was conspicuously hard to the touch but not otherwise painful. Digital examination confirmed our suspicion. High up in the rectum a hard, nodulated, scarcely movable tumor was felt, and, as was observed when the finger was withdrawn covered with blood, an ulcerative tumor.

This made the diagnosis in our case positive. The only question which remained was whether the enlargement of the liver was connected with this carcinoma of the rectum. I believe we must answer this in the affirmative as rectal carcinoma is especially prone to cause metastatic processes by means of the hemorrhoidal veins.

To complement the examination of the tumor we may view the morbid focus directly by means of a speculum which is frequently employed for this purpose. However, I regard digital examination as decisive and usually sufficient. The conditions are different with high-situated tumors which are difficult or impossible to palpate with the finger. Here, as I have convinced myself, the diagnosis may sometimes be made by an examination with the speculum. If this also fails, examination under an anesthetic should never be omitted.

In carcinoma of the rectum in which a tumor can be palpated *examination of the feces* is of secondary importance. Where the tumor is located high, and inspection and palpation give no result, careful and systematic examination of the feces may guide us to a correct diagnosis, from the fact that very frequently or constantly blood, mucus and pus, and occasionally desquamated shreds of tumor, are found. At all events we should not rest until we have satisfactorily explained these very conspicuous changes.

Various deviations from the condition just described may mislead the physician who is not familiar with these symptoms. I can quote a few from my own rich experience without by any means exhausting them. In some patients the disease sets in with decided hemorrhage which, having once occurred, is soon repeated. The patient believes that the bleeding is from a hemorrhoid, and, assisted by popular literature or by his physician, is content with his "golden vein." In other cases marked diarrhea or

actual tenesmus do not point to the rectum but to higher portions of the intestine. In still other instances I have observed how accidental complications such as minor genital affections (in one case uterine retroflexion) so engrossed the attention of the physician that the affection of the rectum was entirely overlooked.

In three of my cases pregnancy was present, and the patient's symptoms were attributed to this until digital examination revealed the existence of a rectal carcinoma. In two of these, fortunately, extirpation was possible.

Simple as the condition appears, the recognition of a carcinoma of the rectum is difficult when the affection has not developed along normal lines, and when it can only be recognized by careful digital examination, which is necessary in every such case of intestinal disturbance.

Cancer of the Small Intestine.—*In carcinoma of the small intestine* we are on much more uncertain ground. Carcinoma of the duodenum shows such similarity to the clinical picture of gastric carcinoma that a differentiation between them is simply impossible. At all events, in carcinomata situated below the papilla of Vater with the constant regurgitation of bilious masses, we may assume a stenosis in the region of the pars descendens duodeni and not beyond this unless, exceptionally, a clearly localized, easily palpable, and otherwise characteristic tumor points to the nature of the affection.

The recognition of so-called *papillary* carcinomata, i. e., those situated and growing around the papilla, is extremely perplexing. Jaundice develops very rapidly, but it is simply impossible to determine its cause on account of the many conditions which are here present. If a tumor can be palpated, sometimes even without this, the diagnosis of a malignant affection may be more or less accurately made from the course of the disease. The determination of its seat is, however, always accidental. In all carcinomata of the duodenum gastric disturbances are the most prominent, particularly vomiting, while symptoms attributed to the intestine are of a secondary nature.

What is true of papillary carcinomata is, in the main, true of the exceedingly rare carcinomata of the *jejunum* and *ileum*, especially as these only exceptionally lead to the formation of a tumor. When a tumor is present it is distinguished by its great movability. Occasionally, when symptoms of stenosis develop with constant vomiting of fecal or fecaloid masses, we may diagnosticate the seat of the tumor as in the jejunum. The appearance of intestinal hemorrhages may then be a further indication of the nature of the disease. The subjective symptoms, in the main, do not differ from those of deeply situated duodenal carcinomata.

DIAGNOSIS

The diagnosis of intestinal carcinomata, as is evident from the foregoing, meets with great and occasionally insurmountable obstacles. We shall not refer to these again, nor shall we enumerate all the errors which may diagnostically come into question, as it is impossible to exhaust the list. We hold that the decisive and most important symptom is the finding of a tumor in the intestine, and even this, aside from the difficulty of its positive localization, does not confer absolute certainty. Thus, for example, a frequent source of error which has lately been much discussed is found in inflammatory tumors of the cecum.

Here, primarily, the differentiation of intestinal carcinomata from tubercular ulcers comes into question.

Obrastzow[1] mentions the following points in the differential diagnosis of these conditions: In carcinoma of the cecum the intestines themselves cannot be palpated, but the tumor with the cylindrical mass attached to it and extending downward as well as the ascending colon passing upward is palpated. In contrast with this, in tuberculosis of the cecum the intestine with its characteristic peculiarities is for the most part susceptible to palpation, but its walls appear thickened and infiltrated. Moreover, palpation usually shows the tumor of carcinoma to have sharply defined borders, while in tuberculosis the infiltration more or less gradually disappears. Finally, in carcinoma of the cecum stenosis very soon occurs.

These points may be unquestionably looked upon as auxiliary factors, but it is doubtful whether in the majority of cases they enable us to reach a positive decision.

The *constant finding of tubercle bacilli* in the feces without simultaneous pulmonary tuberculosis, as mentioned by Obrastzow, is far more decisive. If this is confirmed we will have an additional diagnostic criterion. Besides this, the age, the duration of the disease, the state of the lungs and the course of the fever will aid in the diagnosis. Collectively in isolated cases they make a definite opinion possible, in others this will be tentative, and will remain so until later clinical signs (ascites, etc.) or laparotomy remove all doubts.

Besides ceeal tuberculosis other inflammatory processes from the vermiform appendix, and occasionally also from the typhlon, may assume a tumor-like character so that in deciding as to their nature certain doubts arise. Errors in diagnosis are frequently made in surgical cases from the fact that malignant tumor has been assumed where a simple exudate is present, and *vice versa*. But by careful clinical observation these errors, as a rule, may be avoided.

The same is true of the exceedingly rare cases of benign tumors in the

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[1] *Obrastzow, Arch. f. Verdauungskrankheiten*, Bd. IV, 1898.

cecum (myomata, lipomata, fibromata, etc.), while the differentiation from sarcomata is only possible under very favorable clinical circumstances.

Benign infiltrations may occur in the sigmoid flexure as well as in the cecum (sigmoiditis); occasionally they are of the consistence of a neoplasm and thus make the diagnosis difficult or impossible.

Spastic contractures of the colon must here be referred to; they are frequently associated with obstinate habitual constipation, and are found particularly in the transverse colon, in the cecum, and are occasionally very conspicuous in the descending colon and the sigmoid flexure. As a rule, however, it is possible at once to determine a functional spasm by inflation or by filling the intestine with water. By this means the spasm completely disappears to develop anew after a short time.

We are here upon more positive ground than in the diagnosis of *tumors of the rectum*. In so far as they may be reached by the finger or the speculum, they rarely cause confusion, and above all, *benign neoplasms and syphilitic strictures* here come into question. In regard to the first, with the exception of polypi, which are extremely rare, we observe that benign tumors of the rectum never produce ulceration, while these as a rule occur very early in carcinomata of the rectum; nor does stenosis occur, at least not the very characteristic, rigid stenosis of carcinoma. I must not fail to mention, however, that benign tumors of the rectum, particularly polypi and papillomata, not rarely in later years lay the foundation for carcinoma. I have observed a case of this kind.

Well developed *syphilitic stenoses* present an entirely different appearance from carcinomatous ones. As Kraske[1] correctly states, syphilitic ulcerations never have the coarse, tumorous border of the malignant, and, unlike these, are usually multiple and separated from one another by areas of normal or cicatricially altered mucous membrane. In doubtful cases the diagnosis may also be made by microscopic examination of a small excised particle or a portion that has been detached by the finger.

Diagnosis of Cancer of the Small Intestine.—The diagnosis of carcinoma of the small intestine, even when tumor is present, is most difficult. So long as we are dealing with the upper portion of the duodenum and on account of the identity of the clinical course, the differentiation from gastric carcinoma, as has already been mentioned, is simply accidental. Lower down, particularly in the descending part of the duodenum, the conditions are somewhat more favorable, as the permanent regurgitation of bile is a valuable point in regard to the seat of the tumor. Still further down, however, the clinical symptoms again become indistinct, and only exceptionally permit a well-founded opinion.

The nearer the carcinoma of the small intestine is to the ileo-cecal

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[1] *Kraske*, "Erfahrungen über den Mastdarmkrebs." *Volkmann's Sammlung klin. Vorträge.* 1883–84, p. 789.

valve, the more closely does the clinical picture resemble that of carcinoma of the large intestine, and it becomes impossible positively to diagnosticate the condition.

All this is true of intestinal carcinomata with determinable tumor formation. Where this is absent the conditions, as a rule, are still more complex. Not that the diagnosis under these circumstances is impossible; on the contrary, for example, stenosed carcinomata of the large intestine, even without palpable tumors, may furnish typical cases. In others—at least as far as the determination of a stricture of the large intestine and, proceeding from this fact, a reflection of the entire clinical picture—a probable and even a positive diagnosis of carcinoma of the intestine may be made. Nevertheless, complicating factors frequently mislead the diagnostician, so that great care is necessary.

We waver, however, in our opinion when a palpable tumor and symptoms of stenosis are absent. In this case the clinical impression can at most only indicate the correct direction, and this impression is further strengthened by certain episodes, for example, the onset of severe hemorrhage, the discharge of particles of the tumor, the appearance of palpable metastases, ascites and edema, perforation into neighboring organs which usually occurs at a very late stage, and is unfavorable for "operative" treatment. Examination of the feces may mislead us, especially as the finding of blood and pus is susceptible of many explanations, and does not permit, for example, a differentiation from ulcerative colitis.

COMPLICATIONS AND SEQUELS

The complications and sequels of carcinoma of the intestine in many respects resemble those of carcinoma of the stomach. In one as in the other febrile conditions may appear, usually in the advanced stages of the affection. Coma carcinomatosum has also been observed in cancer of the intestine. Metastasis of the liver, ascites, pleurisy, peritoneal carcinosis, external or internal ruptures occur—particularly into the peritoneum, the bladder and the female genital organs. Other sequels belong to the realm of casuistics, and therefore do not need especial mention at this place.

The chief complication of stenotic carcinoma requires a thorough description: *The transition of partial into complete obstruction.* This may increase slowly and gradually to total occlusion, or it occurs suddenly in a previously permeable intestine and may astonish the patient as well as the physician. The latter may be due to several causes: Either the power of the hypertrophied suprastenotic musculature, which has functioned well, ceases suddenly and completely, or in and immediately above the stricture obstructing fecal plugs or a residue of food have accumulated which, notwithstanding sufficient muscular power, make propulsion impossible, or, finally, a quantity of stagnating fecal masses may produce volvulus.

In these cases a fulminant intestinal occlusion occurs with threatening danger which can only be averted by an early operation. *Pseudo-ileus* may, however, also be present. The patient presents the severe picture of absolute intestinal occlusion. Internal treatment is entirely without result but, nevertheless, contrary to our expectations, the intestines may again become open, the patient may pass gas and feces, and is for the moment saved. But only for the moment! For the next or a succeeding obstruction may become complete and, in spite of medical treatment, pursue its fatal course unless surgery again intervene at the proper time.

PROGNOSIS

The prognosis of cancer of the intestine, as need scarcely be reiterated, is in the main just as grave as that of other carcinomata. But, in the opinion of numerous surgeons, the tendency to the formation of metastasis is not so great as in cancer of the stomach, so that operation performed at the right time offers in the main better chances than in the latter affection. And, as we shall point out in the chapter devoted to treatment, the possibility of total extirpation, for example, in carcinoma of the colon and of the rectum, is more frequent than in carcinoma of the stomach; the circumstance also that in carcinoma of the colon and of the rectum the nutrition is relatively but little affected makes the prognosis somewhat more favorable than in carcinoma situated higher up.

TREATMENT OF CARCINOMATA OF THE STOMACH AND OF THE INTESTINES

This combined description of the treatment of carcinomata of the stomach and intestines has not been without a purpose. The fundamental laws of internal as well as of operative treatment present in both such numerous points of contact that a collective presentation is by far the best to answer didactic requirements.

Complete cure of a gastric or intestinal carcinoma is unknown, not even by means of the knife. The latter by radical or palliative procedures may decidedly prolong life, not, however, or at least very exceptionally, up to the normal, probable duration of life of the individual in question; but even with the highly developed surgical technic of to-day and the popularity of operations on the intestinal canal, this prolongation of life occurs only in a small fractional number of the patients. According to the statistics of G. Heimann[1] made in Prussian hospitals in the years 1885 and 1886, 14.4 and 15.9 per cent. respectively of patients with cancer of the stomach were treated by operation, and even in cancer of the rectum in the same years only 45 per cent., at most 50 per cent., of

[1] *G. Heimann, Arch. f. klin. Chirurgie*, Bd. LVII, p. 4.

the cases were operated upon. We cannot be far wrong in assuming that the remaining number were already in such a hopeless condition that operation appeared to be useless.

Therefore, the majority of patients with cancer of the stomach, even to-day, must be treated by internal means and in no inconsiderable number of those operated upon this is necessary after the operation (palliative or radical).

Internal Treatment.—The objects of *internal treatment* depend in general upon the following considerations: As an actual cure is impossible it becomes necessary, just as in other internal conditions (gall-stones, diabetes) to keep the affection latent as long as possible, and to ameliorate or remove the subjective and objective disturbances which arise. This may suffice for a certain time but permanently it is inadequate, and the reasons for this are manifold.

Primarily, we must consider the mechanical disturbances which frequently arise in gastrointestinal carcinoma, then certain toxic products which are produced by the carcinoma, subsequently the hemorrhages which mediately or immediately are caused by the carcinoma, also metastasis, and, finally, the disturbances of metabolism which, even though not wholly, are nevertheless in great part the consequence of carcinoma.

In regard to the latter, the accurate investigations of F. Kraus, Fr. Müller, and Klemperer, to which those of v. Noorden-Gärtig must be added, have given us full information. They have unanimously shown that a number of patients with cancer, notwithstanding plentiful food and a high amount of albumin, permanently lose N. Fr. Müller found that on increasing the administration of food the conditions remained unchanged. The N-excretion was invariably greater than the N-intake. The increase of acetone observed by F. Blumenthal in severe carcinomatous cachexia points to an increased decomposition of albumin. The more these products increase, the more marked and persistent is the loss of strength, and *vice versa.*

It is, therefore, evident that the nourishment of patients with cancer is extremely difficult. In addition to this, the appetite in the course of carcinoma is very frequently, although fortunately not always, decidedly decreased, chiefly so in carcinoma of the stomach and small intestine, and to a somewhat slighter degree in carcinoma of the colon and rectum.

We must always bear in mind the importance of nourishing patients with cancer as plentifully as possible. Our individual points of view must depend upon the position and the nature of the carcinoma, and the stage in which the patient comes under professional observation.

Let us begin with cancer of the stomach. We find the conditions vastly different according to whether the carcinoma runs its course with or without very decided motor disturbance, or, *vice versa*, severe symptoms of stagnation are present; in the latter case, as a rule, vomiting

dominates the scene. In the former case, a decided limitation of diet is not indicated, or to but very slight extent. Albumin, carbohydrates, and fat, naturally most carefully prepared, are permissible, and above all milk and milk preparations in the most varied form and according to the individual circumstances and the wishes of the patient.

With a fairly good appetite and the careful combination of foods, it may be possible to keep the patient for weeks and months in good condition, and now and then even to increase his weight. Occasionally the patients are subjectively better, and their courage revives, a fact to which the gain in weight conduces not a little.

It is unfortunate that most patients with cancer of the stomach have a distinct repugnance to meat and meat derivatives (therefore to bouillon and meat extracts). Hence the administration of meat cannot be insisted upon, but an attempt should be made to administer other albumin bodies, with eggs, carbohydrates rich in albumin, dishes prepared with milk, etc. After a prolonged abstinence from meat an attempt may be made again to administer it in some form, but usually without success.

The nourishment of the patient is much more difficult in carcinoma at or near the pylorus. Here, as a rule, we are limited to fluid food, and often but small quantities will pass through the pylorus. Above all, milk preparations, soup with the yolk of an egg, the various leguminous vegetables, and cereals in convenient form, may prove serviceable. That only small quantities should be administered will be understood. From the quantity of urine voided, and by comparing the amount of material found in the stomach by lavage in the early morning with the amount of fluid introduced, we can practically estimate the amount absorbed.

It is perhaps well at this point to touch upon the value of *artificial food preparations* in cancer of the stomach. We may to-day regard it as certain that they have no beneficial effect on the nutrition.

Their value consists solely in the fact that the patients who have no appetite for food, but actual repugnance, regard the intake of these substances mixed with nutritive preparations as a duty to which they submit as willingly as to that of taking medicine. From this standpoint it is quite immaterial what artificial food we administer to cancer patients. The individual taste will best decide. According to my personal experience the most suitable preparations are: Puro, meat jelly, somatose, sanatogen, roborat. These products will also furnish a sufficient variety.

Luxuries, substances *which tempt and stimulate the appetite* (v. Leyden), are just as important as nutritive preparations. What has been said above is also true of these. By their aid it is often possible for a long time to keep the patient away from the cliffs of inanition. What is advisable in the individual case depends upon the social position, upon the habits, and finally upon the wishes of the patient. At all events by being too strict we may do more harm than good.

Among the auxiliary dietetic measures belong *artificial,* or, as it has lately been called, *extra-buccal* nutrition. For the technic and other details we must refer to von Leube's excellent description. It only remains for us briefly to mention the value of this in carcinoma of the stomach. Unquestionably the mere introduction of fluid to prevent a too dry condition of the organism may also prevent threatening tetany and the accumulation of toxic products, and the intake of even a few calories is a gain which should never be undervalued.

No one will claim that extra-buccal nutrition decidedly prolongs the life of the patient with cancer. The simplest method and the one most in use is by means of *nutritive enemata*; subcutaneous methods (oil-sugar solutions), no matter upon what scientific principle they are based, have, as yet, not found general acceptance.

What is true of cancer of the stomach is also true of *carcinoma of the small intestine.*

In carcinoma of the large intestine other points of view arise, and, above all, it is here an absolute law that we should avoid everything in diet which may obstruct or delay the passage of the feces. *Avoidance of all substances with skins containing residue unsuitable to the gastric and intestinal juices* is therefore a leading principle. Then the nutrition must be calculated directly to stimulate intestinal peristalsis. This may be attained by *physiologic aperients*: Honey, milk sugar, sour milk, stewed fruit, lemonade, wines made from fruit, marmalade, and fruit jellies. Other foods may be given in the form and manner mentioned under the consideration of cancer of the stomach.

In ulcerating carcinomata which do not run their course with the formation of stenosis, we must be particularly careful of the diet. The most suitable articles here are milk and milk preparations, soups made from leguminous vegetables, soups containing eggs, meat jelly, etc. In extreme stenosis from obstruction the introduction of food is most difficult on account of the mechanical hindrance to the passage of feces. Under these circumstances we must limit ourselves to small amounts of highly concentrated food.

The artificial introduction of food in carcinoma of the large intestine is only permissible provided the neoplasm does not extend lower down than the cecum or the flexura colico-hepatica. Below this it will irritate rather than benefit the nutrition. In carcinoma of the sigmoid flexure there can naturally be no question of rectal alimentation. Reports in regard to subcutaneous nutrition, which is here indicated, are not to my knowledge at hand.

Drug Treatment.—To ameliorate the sufferings which carcinomata of the stomach and intestines bring in their train drugs are often useful, occasionally even indispensable.

One of the most frequent causes of distress in gastric and occasionally

also in intestinal carcinoma is *loss of appetite*. Often this can be relieved for a time by the so-called stomachics, by condurango as well as by other bitters; as a rule, however, they fail. In so-called achylia gastrica, which, as before stated, very frequently occurs in gastric cancer, we may employ hydrochloric acid and also pancreatic preparations to improve the digestion. Recently pankreon has been much employed in the form of tablets each containing one gram.

Pain in the region of the stomach and particularly of the intestine also calls for relief. In gastric pain, as a rule, mild narcotics (codein, dionin) suffice. In severe cases of obstruction, in intestinal carcinoma, the most powerful narcotics are necessary. First among these is opium in the form of the tincture of thebain, or pure opium (0.03–0.05) perhaps in combination with belladonna (extract of belladonna 0.01–0.03), then morphin as a powder, in solution, by suppositories or subcutaneously.

Obstinate constipation by no means contraindicates the administration of opium; on the contrary we frequently see that after complete intestinal rest normal intestinal peristalsis again sets in.

Opiates act remarkably well in the severe rectal and vesical tenesmus which we have learned to recognize as regular accompaniments of deep-seated carcinoma. By the influence of these drugs the patients may be spared these distressing symptoms for many hours during the day or during the night.

Purgatives play an important rôle in the treatment of gastric and intestinal carcinomata. In the former purgatives will only rarely be administered, preference being given to lavage. If aperients are indicated, fluids or substances readily soluble in water are preferable to pills and also to tablets which are frequently dissolved with difficulty or not at all.

In the stage of intestinal rest in carcinoma of the intestine, purgatives are usually indispensable, and here also purgatives that are easily soluble yet at the same time are mild and act quickly are the best. As most suitable remedies for this purpose I advise: Fluid extract of cascara, compound licorice powder, castor oil, magnesium, rhubarb, flowers of sulphur and, finally, mineral waters in small doses. In intestinal carcinoma it is wise to keep the bowels regulated by the continuous use of a suitable purgative and thus avoid being surprised by the sudden cessation of peristaltic action.

Mechanical Treatment (*gastric lavage*).—Gastric lavage in cancer of the stomach and small intestine may prove useful and palliative, but even here we must individualize strictly. This should not be resorted to so long as the motor activity of the stomach is unimpaired. It is true that lavage acts favorably upon the pain, also occasionally, when stagnation exists, upon the appetite.

But even when this is the case, the nature of the carcinoma, its stage,

and the strength of the patient must be carefully considered. In stagnation not too far advanced lavage may be employed, provided pain, a sensation of pressure, nausea, vomiting and anorexia are present. Stagnation itself, in my experience, does not necessitate the employment of lavage. In the course of years I have seen numerous patients with cancer of the stomach who, in spite of moderate stagnation, remained in fair condition for quite a long time.

With severe stagnation and coffee-ground contents I no longer employ gastric lavage, because I have observed that the patients are much worse after this treatment than before. If it be necessary to remove the stagnant masses, an expression limited to a few minutes is sufficient. But even in incipient cases I believe it wise to resort to gastric lavage as little as possible. With care in the diet we are frequently able to prevent abnormal collections of material.

Indications for, and Results of, Operative Treatment.—The physician is to-day in duty bound carefully to study the indications for, and the results of, abdominal surgery. In advising or rejecting operation, he is often held responsible both for what is done and what remains undone. He will base his opinion, in the first place, upon statistics of large groups of cases, and then upon personal experience. In any event, no matter how the individual case may present itself, the following questions are sure to arise:

1. Is surgical interference indicated in the stage in which this patient presents himself?

2. If this be the case, what operation is indicated, and what are the probabilities of success?

In some individuals the answer to the first question is easy, in others difficult or even impossible. The decision may be quickly made in dealing with very cachectic individuals, also in those cases in which diffused edema, ascites, diabetes, hepatic metastases, or serious complications on the part of the heart, the lungs or the kidneys exist. Under these circumstances, naturally, any surgical interference will only hasten the end. Nor will we resort to operation if, for example, there is a very circumscribed carcinoma of the small curvature, which neither subjectively causes severe symptoms, nor objectively produces serious motor disturbance. Experience teaches us that in such cases the duration of the patient's life is no less than after operation, provided that exceptionally a radical removal is indicated by the position of the growth. According to my experience the prognosis is very serious in those cases of carcinoma (usually at the pylorus) in which the vomited material or the evacuated gastric contents resemble coffee-grounds. The results which I have seen after even palliative operation can scarcely be called good.

In carcinoma of the large intestine and rectum the conditions are quite similar, but the lower down the growth is situated, the more favor-

able are the prospects of a radical operation. We shall revert to this later.

In regard to the second question, if there are no contra-indications such as have been mentioned, surgical interference becomes necessary and we must now consider the question: What operation is to be performed, shall it be the radical removal of the tumor or merely palliative treatment, i. e., the removal of the motor disturbance?

Let us begin with the *stomach*. In the first place, we must confess that an absolutely positive decision as to whether extirpation or gastro-enterostomy is indicated is most difficult to answer *prior* to the laparotomy. No matter how favorable the location of the tumor, laparotomy may show such a general extension of the carcinoma that a radical removal of the tumor is simply impossible. v. Mikuliez and Kausch[1] therefore very properly remark that every operation is primarily an exploratory laparotomy, and a definite plan of operation depends upon what is found on opening the abdominal cavity.

In many cases we may at once exclude the possibility of total extirpation, for example, when the tumors are large and immovable or when the tumor is movable but the patient has not sufficient strength.

In non-palpable tumors the decision is still less possible prior to exploratory incision. When we have reason to assume that a pylorie carcinoma causes stenosis, a radical operation is at least possible. In other cases, however, even with a positive diagnosis, such an operation can hardly be advised.

Besides gastroenterostomy, as a palliative proeedure we should also consider *jejunostomy* which is particularly advised by v. Maydl. He introduced this operation for the cases of gastrie disease in which a great portion of the stomach was destroyed, and in which it was impossible to supply sufficient nutrition.

Recently this surgeon has advised us to employ jejunostomy in place of gastroenterostomy, as the former is much more thorough, and, besides, meets the important requirements of the case by sparing the diseased organ and protecting the neoplasm from all irritation.

However, this operation has been employed by surgeons only to a limited extent, chiefly for the reason that, like all operations for fistula, cosmetically it is not fully satisfactory. The prolongation of life, too, is less than after gastroenterostomy.

As a very thorough operation, total extirpation of the stomach, the union of the upper duodenum and the cardia, or the lowest portion of the esophagus, which was first successfully performed by Schlatter in Zurich, comes into question. Although to-day quite a number of favor-

[1] *v. Mikuliez* und *Kausch*, "Handbuch der praktischen Chirurgie," Separatabdruck, p. 147.

able results are reported in surgical literature, nevertheless, as v. Mikulicz and Kausch [1] state, metastatic lymph-glands are rarely absent in any case of gastric carcinoma, and, therefore, we should not indulge in too great expectations of the results of this heroic treatment. On the other hand, it must be admitted that some remarkable successes—for instance, the last case operated upon by Fedor Krause—favor the radical removal of the neoplasm. At the present time, however, the definite results, the failures and successes, cannot be positively recorded.

In the main, *gastroenterostomy* and *resection of the pylorus* are the operations at this time most frequently performed.

With the results of these methods we are comparatively well acquainted, a large number of statistics being available, as well as the individual reports of eminent surgeons. Thus, Terrier and Hartmann have collected the statistics of abdominal operations performed by distinguished surgeons (Czerny, Krönlein, Carle, v. Mikulicz, Kocher, and Hartmann) and from 127 resections of the pylorus have calculated a mortality of 26 per cent.; but this varies greatly. For example, Lindner has lately estimated the mortality at 50 per cent. In the main, however, the results of resection in the course of the last twenty years have gradually improved. v. Mikulicz [2] computed the total of radical cures at 17 per cent.

If this estimate is compared with the total number of cures of other carcinomata we are by no means hopeless. According to v. Mikulicz, the cases of carcinoma of the breast permanently cured amount to from 10 to 15 per cent., with curettage of the axillary cavity to 25 to 30 per cent., in carcinoma of the uterus 30 to 35 per cent., in carcinoma of the rectum 10 to 20 per cent., in carcinoma of the tongue 10 to 20 per cent. If we compare these with the results of gastroenterostomy, the latter, on account of a more complete technic, and, above all, by the avoidance of the so-called vicious circle, are decidedly better. While, according to the last great statistical report of Chlumky, the mortality from 1881 to 1885 was still 75 per cent., from 1886 to 1890 it decreased to 48.4 per cent., and from 1890 to 1896 declined to 36.61 per cent.; for the past five years v. Mikulicz estimated his operative loss at only 28 per cent. (in his last report v. Mikulicz computes these fatalities at 26 per cent.).

In gastroenterostomy, however, not only the immediate results of the operation but the prolongation of life as well as the functional results must also be considered. As to the former, the average results are not very satisfactory. Life was prolonged for five or six months; now and then there must have been brilliant exceptions to this.

Nor is the functional effect in all cases satisfactory. Frequently the

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[1] *L. c.*

[2] *v. Mikulicz, 73. Versammlung deutscher Naturforscher und Aerzte in Hamburg,* 1901. Referat.

pains and vomiting recur, jaundice, ascites and edema appear, and a persistent anorexia leads to early loss of strength. Even in favorable cases, in spite of an increase in weight, the patients do not gain in strength, and are unable to follow their usual occupations. *In other words, the prolongation of life is frequently nothing more than a prolongation of suffering.* It is true some strikingly favorable results occur, but in the overwhelming majority of cases, after a brief improvement a progressive downward course is noted.

It follows from this that, although the immediate results of gastroenterostomy are relatively beneficial, for the duration of life as well as for the general bodily condition and functions, after gastroenterostomy there is still much to be desired.

Under these circumstances surgeons as well as physicians frequently raise the question whether gastroenterostomy should not be limited in favor of total extirpation. After I had formulated this conclusion based upon my own experience,[1] v. Mikulicz, in the article cited above, and from his far greater experience, arrived at the same opinion.

If from this standpoint we make a strict choice, the number of cases suitable for resection will not be great, perhaps will even be less than before, but the patient will have the benefit of not only an apparent but an actual result which may in some cases prolong life for many years.

At the present time, our conclusions amount to this, that in cases in which total extirpation cannot be performed we should under special circumstances advise gastroenterostomy; for example, when there are very marked disturbances (pain, vomiting), a fair degree of strength, and when the use of internal remedies has been futile. Occasionally, at the urgent desire of the patient and his relatives, to whom, for personal reasons, even a brief prolongation of life is a boon, we are constrained to consent. When the question of total extirpation arises, and the surgeon considers the patient's strength sufficient, this operation should be performed. Often enough, in such cases, we will be convinced of the futility of total extirpation and content ourselves with gastroenterostomy, but now and then the constellation of conditions appears so favorable that radical procedures are undertaken.

We now turn to *carcinoma of the intestines*, and in the case of carcinoma of the small intestine the laws and indications are the same as those which have just been described. The higher the situation of the carcinoma the more difficult is its total removal, as the duodenum normally is so fixed that the total extirpation of a tumor is not only extremely difficult and attended therefore with great loss of time but is combined with great danger for the patient. Lower down, as is proven by a few successful sarcoma operations, complete success has been attained. But

[1] *Boas*, "Diagnostik und Therapie der Magenkrankheiten," II, 4, Auflage, p. 222.

the decision as to the most suitable time for operation meets with many obstacles.

The conditions are more favorable in carcinomata of the large intestine and rectum. Whenever possible the radical method is here to be preferred to the palliative. Generally speaking, the conditions for total extirpation in the previously discussed carcinoma varieties are decidedly better than in carcinoma of the stomach; for, as we have stated, metastases usually occur late, the tumor being for a long time movable, and less technical difficulty is encountered at the operation.

In spite of this the results fall far below our expectations. According to Wölffler-Schloffer, up to the year 1896 the mortality in resections of carcinoma of the large intestine still amounted to 50 per cent. Yet the prognosis in regard to prolongation of life appears to be decidedly better than in high-seated carcinomata. Cases are on record of cure lasting seventeen years (Martini-Gussenbauer), of ten years (Czerny, a case of sarcoma), of eight and nine years (Körte), etc. I have a patient who had carcinoma of the cecum (adenocarcinoma) and whose recovery has lasted over five years.

Nevertheless, up to the present time such recoveries are rare. Most cases, even after a radical operation, perish in the next two years.

In case a radical operation cannot be performed two palliative methods of treatment remain: Entero-anastomosis and anus præter naturam (colostomy). Unquestionably the first method is preferable to the other for obvious reasons. It also gives relatively good results—according to Wölffler-Schloffer the mortality is 20 per cent.—and prolongs life from a year to a year and a half. But here we must bear in mind not only that the patients live, but how they live. Although there are very favorable functional results, I have repeatedly observed, soon after the entero-anastomosis, renewed attacks of severe colic, marked loss of weight which had at first been increased, edema, and ascites.

In conclusion, *colostomy* will only come into question when, on account of the general condition, an immediate operation is necessary, therefore either when the strength is markedly reduced in the early stages of obstruction, or where debility has more fully developed. These conditions will usually be found associated. The indications for colostomy are closely related to the situation of the carcinoma. For example, in carcinoma of the cecum the production of a fistula of the ileum is a questionable proceeding, both from a cosmetic and a nutritive standpoint. The true domain of colostomy is found in carcinoma of the sigmoid flexure and of the rectum, for here the feces have acquired a consistence which makes it possible readily to remove them.

We now turn to the operative treatment of *carcinoma of the rectum*. According to the seat, the development, the movability of the tumor, and the presence of metastases, the following procedures must be considered:

Extirpation of the tumor, curettage, and the production of an artificial anus.

The ideal method is, naturally, the radical removal of the tumor (amputatio recti or resectio recti), but, only when the tumor is sufficiently movable, and when there are no metastases of distant organs, especially of the liver, is this possible. Enlargement of regional lymph-glands, however, forms no contra-indication to total extirpation. Since the performance of resection of the sacrum by Kraske (1882) high-seated carcinomata of the rectum have also been radically removed.

The results of these operations at the hands of different surgeons are far asunder, but in the last ten years they have become decidedly more favorable. According to Czerny's comprehensive statistics of the Heidelberg Clinic, which have the advantage of originating with him and which cover a long period of time, 152 rectal carcinomata were observed in the years from 1878 to 1891 and 109 radical operations were performed. Among 83 cases which were operated upon by the perineal method 3 died, = 3.6 per cent.; of 66 according to the sacral method, 9 perished, giving 13.64 per cent. The total mortality of 10 deaths in 109 cases is 9.1 per cent. Of 99 in whom radical operation was performed followed by a cure, 21 lived two years and more after the operation, 15, three years and longer, 13, four years and longer, 8, five years and longer; among these one case lived for 18 years, one case for 16 years, four cases respectively thirteen years and nine months, eleven years and six months, eight years and nine months, and six years and nine months. The frequency of relapse after extirpation is calculated differently by different authors. The figures vary between 41.6 and 73.3 per cent. Czerny maintains that 20 to 25 per cent. of those in whom radical operation is performed continue free from relapses for two years, and the majority of them are permanently cured. The sacral method in particular diminishes the danger of relapse, as the lymph-glands situated in the excavatio sacralis are likewise removed.

The functional results vary according to whether the sphincter is retained or removed. In the former case, the functional results, *quoad continentiam recti,* are very satisfactory. In the latter, the condition of the patient is most unpleasant, as he can retain neither gas nor fluid feces, and it is therefore necessary by diet and drugs to make the feces compact.

Where a radical operation is impossible, as is unfortunately often the case, there are two methods, curetting and the production of an artificial anus—the one direct, the other indirect—by which we diminish or arrest the symptoms of stenosis. The curetting which may be performed with instruments, or, still better, with the hands, is best adapted to deep-seated carcinomata with special implication of the posterior wall of the rectum. When the tumor is high-seated it is best not to use this method as, under

some circumstances, more harm than good is done. The same is true of deep-seated carcinomata in the anterior wall of the rectum.

The most suitable and, relatively, the least dangerous measure for the removal of the symptoms of stenosis is unquestionably colostomy. This method most certainly relieves the irritation of the tumor, and cases are on record of prolongation of life for two or three years after the production of an artificial anus.

In regard to the indications for colostomy the opinions of physicians and surgeons are wide asunder. While colostomy is frequently employed in England and France, in Germany, like all fistulous operations, it appears to have lost favor in the last few years. We may here speak of a relative and an absolute indication for its performance. The indication is relative if, while a fair intestinal passage remains, the patient steadily loses in weight and strength, is tormented by pain, and passes much pus and blood. In such cases we are decidedly in favor of operation, provided internal remedies have proven ineffectual. As long as evacuations at all sufficient are brought about by mild dietetic measures or by purgatives, we do not advise colostomy. With a suitable diet I have kept well advanced cases of carcinoma of the rectum alive just as long as by the production of an artificial anus.

Colostomy is absolutely indicated when complete intestinal occlusion occurs or is threatened. In this case it is best not to wait so long that the patient's strength is diminished by vomiting, pain, and high-graded meteorism. The functional results of colostomy are satisfactory, provided we are careful to produce compact feces and the fistulous opening is kept well closed with a hernia bandage. We must consider, too, that many patients naturally find the daily manipulation of their intestines and of the dejecta exceedingly repugnant. It is, therefore, the duty of the physician and the surgeon, in order to prevent subsequent regret, to explain to the patient in no uncertain manner before the operation the light and shade aspects of the anus præternaturalis.

DISPLACEMENTS OF THE ABDOMINAL VISCERA AND OF THE HEART

By F. HIRSCHFELD, Berlin.

INTRODUCTION

Until about twenty years ago a displacement of the viscera was generally regarded as a rarity. It is true that at the beginning of the nineteenth century celebrated clinicians such as Esquirol had devoted much consideration to displacements of the abdominal viscera, and had even recognized the connection between displacement of the colon and the development of mental disease; but, toward the middle of the century, these views were no longer considered tenable, as appears from the works of Griesinger, Wunderlich, Canstatt and others. Displacement of the colon was looked upon as unimportant, that of the stomach could not be detected by the ordinary clinical methods of the day, and only that of the kidneys was considered, this, however, being regarded as a rare occurrence since it was only occasionally noted by anatomists. The reason for this may be found in the technic of autopsies, as well as in the fact that in most cadavers the organs have regained their normal position by the bodily rest which usually precedes death. It is true that in isolated reports abnormal movability was occasionally pointed out, but general attention was directed to this only about in 1880, in Germany, by the labors of Landau, Leube, Ewald, L. Kuttner, Litten and Meinert, and in France by Glénard, Féréol and Cuillerct. In 1890, Virchow demonstrated that a change in the position of the abdominal viscera, and particularly of the intestines, could be detected in the majority of persons. Glénard did much to promote the recognition of these changes in position as a pathologic condition by creating the name, *enteroptosis*. His investigations, however, soon led him to a path on which he could no longer be followed. His original view that enteroptosis is a sharply characterized substantive disease (entité morbide) was not at once generally accepted, but in the main was considered justifiable. It was admitted that in many persons a moderate degree of downward displacement of the stomach, a unilateral movability of the kidney, and a displacement of the colon could be demonstrated,

and the embodiment of these phenomena in one clinical picture appeared all the more warranted since they presented themselves in nervous, weak females, usually anemic, therefore in persons who in their external appearance already presented the same type. Glénard believed that downward displacement of the colon, particularly of the right portion of the transverse colon, was chiefly responsible for the production of these disturbances. On the other hand, in the development of displacement of these organs, he was but little inclined to take into consideration mechanical conditions, flaccidity of the abdominal walls, the disappearance of fat in the abdominal spaces, the influence of external pressure from lacing, etc. The importance of these factors in the etiology of enteroptosis was especially demonstrated by Landau, Meinert, and Dennig, and was proven by the fact that in many persons, and under certain conditions, a loosening of the attachments and the descent of one or more organs was possible. The anatomical prior condition may be favorable to a displacement in one case, still more so in another, but it is difficult to regard this weakness of the ligaments as the foundation of a disease, particularly as it occurs in most persons without producing symptoms. Thus we see floating kidney appear in one person by the development of asthma and emphysema, because of the disappearance of fat in the abdominal cavity, and, perhaps, because the flaccidity of the abdominal walls which increases with age diminishes pressure in the abdomen, and thus the firm position of the kidneys is so far weakened that under the influence of shock to the abdominal cavity (as in prolonged paroxysms of coughing) they finally descend; in other cases, however, we note particularly how the heart is limited in its movability by the formation of empyema in the lungs, and subsequently the phenomenon of "displaced heart" disappears. Only the minute consideration of all the mechanical conditions will enable us to understand clearly the genesis of these changes in position and their influence upon the functions of the various organs.

By some clinicians, such as Stiller and Obrastzow, enteroptosis or splanchnoptosis is to a certain extent regarded as the sign of degeneration. The imperfect cartilaginous attachment of the tenth rib to the thorax (costa decima fluctuans), frequently found in such persons, is looked upon as proof. A certain justification must be admitted for this view, since, undoubtedly, a displacement of the abdominal viscera is found in a greater percentage of persons predisposed to nervous affections than in the normally strong. On the other hand, we must bear in mind that in nervous persons a lessened muscular activity, a weaker muscular structure, and probably a more flaccid ligamentous apparatus, are generally to be expected. Therefore, that a displacement of the internal organs may readily occur does not appear remarkable, and this circumstance naturally explains the greater distribution of enteroptosis among the nervous. Whether, in addition, a certain, and perhaps also a hereditary, predisposition can be as-

sumed is extremely difficult to decide. Very likely a feeble ligamentous apparatus in the internal organs and certain peculiarities in the structure of the same may be hereditary, as well as a special predisposition of the bony skeleton; but, for the development of enteroptosis, perhaps, just as in the development of kyphoscoliosis of the vertebral column, a number of external influences are necessary. Therefore, we do not agree with Glénard who, in accepting the French views regarding l'arthritisme, propounds a theory according to which most diseases are related to enteroptosis, and a disturbance in the activity of the liver which is not described (l'hépatisme) is the most significant symptom.[1]

Although certain uniform factors are decisive in the development and treatment of the various displacements of the organs, it appears to be most necessary for the correct understanding of the subject under discussion to describe the displacement of each individual organ separately.

Most important are the displacements of the stomach and the kidneys. Displacements of the spleen and of the liver are generally rare, those of the colon and heart more frequent but of less clinical importance.

DISPLACEMENT OF THE STOMACH (GASTROPTOSIS)

Reports vary as to the frequency of gastroptosis. According to Meinert,[2] in Dresden the majority of women have a displacement of the stomach while, by estimation, only about 5 per cent. of men show an alteration in the position of this organ. Similar proportions have been reported by Dennig[3] in the population of Württemberg. From his figures it is very evident in which years of life gastroptosis is specially prone to appear.

Among 2,000 persons who presented themselves at a Stuttgart Polyclinic on account of various ailments not connected with disease of the stomach,

in 29.7 per cent. of the men,
in 75.4 per cent. of the women,

a displacement of the stomach was determined.

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[1] The views of Glénard, except those in his earlier publications (*Lyon médical*, 1885 et 1887), are found in his comprehensive work: "Les ptoses viscérales. Diagnostic et nosographie." Paris, 1899, 962 pages. Compare also Le progrès médical, 1899, I, page 320; 1900, I, page 225; 1902, I, No. 2.

[2] *Meinert, Sammlung klin. Vorträge.* N. F., Nr. 115 u. 116, Leipzig, 1895; *Centralblatt f. innere Med.*, 1895, Nr. 43 und ebenda, 1896, Nr. 13 u. 14.

[3] *A. Dennig, Württembergisches med. Correspondenzbl.*, 1903, Nr. 18.

In regard to age, gastroptosis was present

	In Men.	In Women.
	Per cent.	Per cent.
From 14–20 years of age in	16	..
" 21–25 " "	27	62
" 26–30 " "	26	79
" 31–35 " "	28	81
" 36–40 " "	37	92
" 41–45 " "	29	96
" 46–50 " "	38	92
" 51–55 " "	34	85
" 56–60 " "	34	97
" 61–70 " "	32	100

It was determined in girls

from 10–15 years, in 20 per cent.,
" 16–20 " " 57 "

From this it can be distinctly seen that gastroptosis is much more frequent in females than in males. This displacement probably occurs even at the age of puberty as a consequence of wearing a corset, and under the influence of pregnancy in the following decades flaccidity of the abdominal walls becomes more common until, finally, almost all elderly women show gastroptosis. According to Dennig the same deleterious influence as that of the corset is ascribed to a habit very common among the female rural population of Württemberg of tying the skirts around the waist and drawing the strings very tight. When the hips have not as yet attained the growth characteristic in woman, tightness about the waist is especially desired to prevent the skirt from slipping down. Suspension of the clothes from the shoulder, as is the rule with children, is seldom seen in girls over fifteen years of age.

The correctness of these conclusions is proven by the results of investigations in males. Here the injurious effects of wearing a belt to fasten the trousers instead of suspenders from the shoulder is distinctly recognized, for of 172 men with marked gastroptosis, 72 wore narrow belts around the waist, while among 406 men with a normal position of the stomach only 16 used the belt; the remainder wore suspenders.

In children, as a rule, the stomach lies in a horizontal position. Meinert declares it to be true that every child is born with a vertically lying stomach, the pylorus of which is therefore deeply situated, but that after a few weeks this position is changed. The lower curvature of the stomach forms an almost horizontal line above the umbilicus.

The conspicuously frequent occurrence of gastroptosis in Saxony, which was first pointed out by Meinert, has given rise to much speculation. Some investigators reject the entire theory, and believe that the large

amounts of gases developed from the carbonic acid employed for distending the stomach abnormally displace this organ, and probably also give no well-defined idea of the size of the stomach so inflated. According to Meinert, in the above investigations

8 grams of sodium bicarbonate,
6 " of tartaric acid,

were employed; this was dissolved in a little water and taken at once or in divided doses rapidly succeeding each other. Gastroptosis was assumed whenever the pylorus could not be recognized at the serobiculus cordis, or was situated more deeply; the greater and lesser curvatures of the stomach must have descended at the same time.

As large doses of this mixture will produce nearly 3 liters of carbonic acid, the criticism is justifiable that the normal stomach which, as a rule, has merely a capacity of about $1\frac{1}{2}$ liters, is immoderately distended and perhaps also is drawn somewhat downward. Dennig employed a large double bellows with which he inflated the stomach after introducing the stomach-tube. With this method, as I have fully convinced myself, particularly with tense belly walls, it is necessary to exert considerable pressure if the boundaries of the stomach are to be sharply defined. We must, however, remember that neither in powerful men nor in children does this downward displacement of the distended stomach ever take place. We are thus forced to conclude *that this property of the stomach or, rather, of the pylorus, to descend upon immoderate inflation,* is a pathologic deviation, and to a certain extent indicates a predisposition to gastroptosis.

In the description of the development of gastroptosis from the influence of gastric catarrh, general weakness of the stomach, etc., I shall attempt to show that, to a certain extent, the same process takes place as in the downward displacement of the organ artificially inflated by carbonic acid gas.

The purely mechanical course may best be understood by some illustrations.[1]

In Fig. 11 we see a stomach in its normal position. The pylorus is situated at a point almost directly behind the right arch of the ribs, where this is crossed by the tip of the ensiform process upon a horizontal plane (Meinert). The axis of the stomach more nearly approaches the horizontal than the vertical. The pyloric portion of the stomach is in the main situated in the serobiculus cordis.

In Fig. 12 we see the so-called vertical position of the large normal

[1] These illustrations have been taken from the "Diagnostic Lexicon" of *A. Baum* and *Schnirer*, Vienna, 1884, III, p. 111. Presentation of gastric examination by Rosenheim. My description of the individual figures differs somewhat from that given by Rosenheim.

stomach when inflated. The pylorus can no longer be detected in the pit of the stomach. The greater curvature is decidedly lower than the umbilicus. The axis of the stomach is in a vertical rather than a horizontal position.

Fig. 13 shows a somewhat later stage. The greater curvature has descended lower. The cardia may be recognized in about the same area as

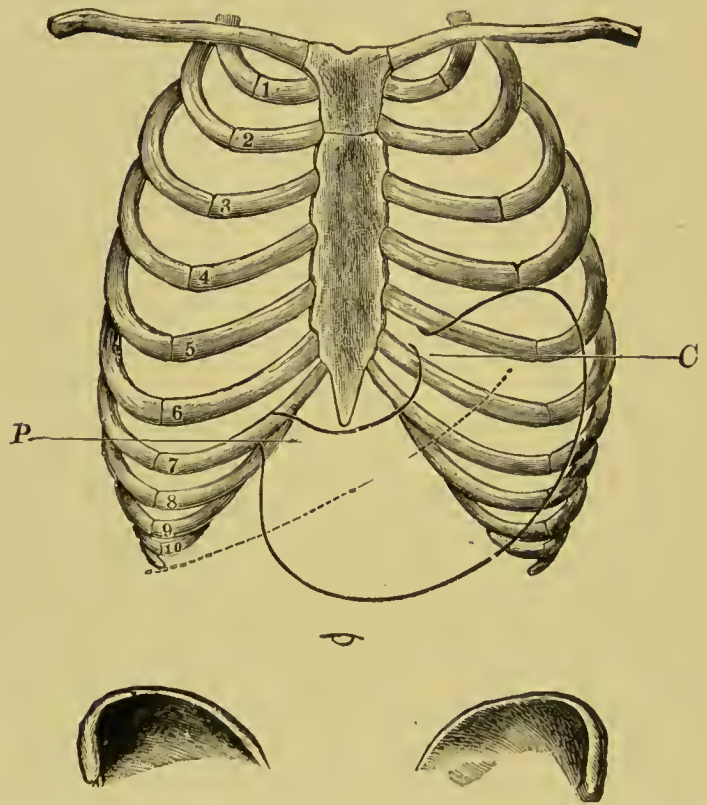

FIG. 11.—Normal situation of the stomach; dotted line, ..., shows the margin of the liver.

in the normal stomach. Especially noteworthy is the great bulging of the pyloric portion of the stomach downward and to the right. As the muscles of the stomach which chiefly propel the chyle are situated around the pylorus, an enlargement of the stomach to the right, which may be distinctly demonstrated by percussion, clearly denotes a weakness of the muscular apparatus and beginning insufficiency of the same,[1] although gastric dulness appears, in the main, to be but slightly increased.

In Fig. 14 we see a decidedly enlarged stomach. Such a condition may have been caused by gastroptosis and subsequent insufficiency as well as by a mere descent from gastrectasis. A decision cannot be based alone upon these pictures; for this purpose the consideration of all the points necessary in an examination is called for.

[1] *Rosenheim*, "Krankheiten der Speiseröhre und des Magens," I. Aufl. 1891; Leo, *Deutsch. med. Wochenschr.* 1896; und *W. Michaelis, Zeitschr. f. klin. Med.*, XXXIV.

A somewhat different condition is shown by Fig. 15, which is taken from Bollinger's book.[1]

This illustration of Bollinger, first of all, shows distinctly how the vertical position of the stomach with constriction of its pyloric portion occurs from the external pressure of tight lacing, just as this acts upon the liver. A vertical position of the stomach without dilatation is frequently not considered pathologic by the anatomist, and therefore we often see illustrations of a vertical stomach in books upon normal topographic anatomy.

How do the effects of a downward displacement of the pylorus, of gastroptosis, become noticeable during life?

In a normal person this occurs by downward pressure on the liver and pylorus in consequence of tight lacing around the waist, and, except for slight discomfort which, perhaps, is only due to pressure upon the skin, the activity of these organs is usually but slightly affected. It is true

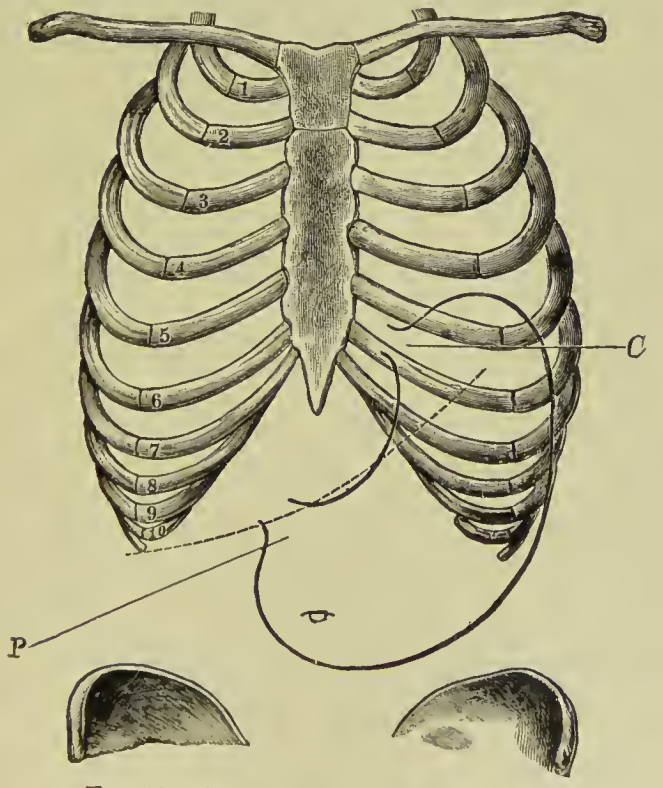

FIG. 12.—Gastroptosis of the first degree.

the passage of the chyle from the stomach into the duodenum is probably impeded, but we know that the smooth muscles of the intestine rapidly hypertrophy after the addition of obstructions in the intestinal canal, and thus by increased labor bring about compensation. The majority of healthy

[1] *O. Bollinger*, "Atlas und Grundriss der pathologischen Anatomie," I., page 193, 2. Aufl. München, 1901.

young girls, therefore, notice no immediate, injurious effect from the wearing of a constricting corset which simultaneously presses down the liver and the pylorus. It is different, however, if the stomach of the

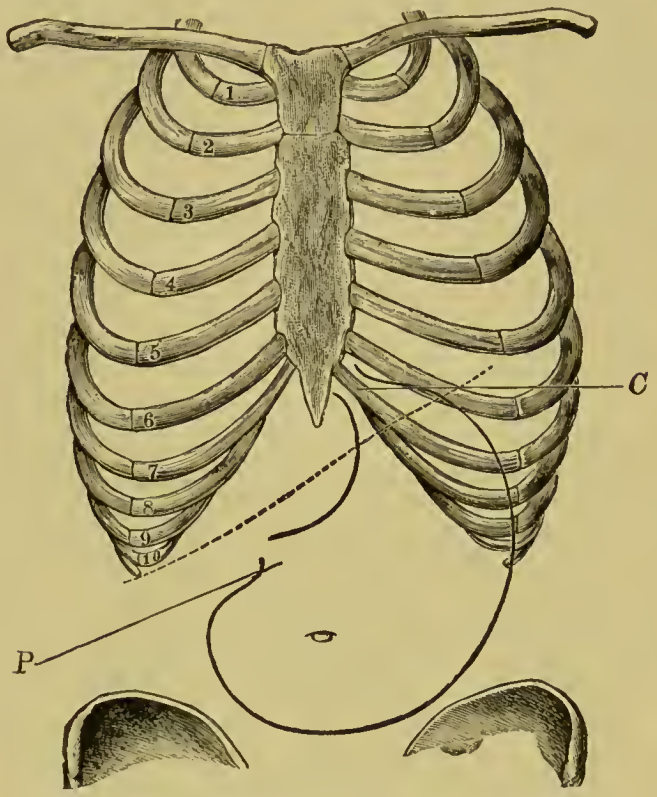

FIG. 13.—Gastroptosis of the second degree.

person in question has previously shown a tendency to disturbance, or if a general weakness of the system exists.

These disturbances most frequently occur in young, anemic, poorly nourished, growing girls. Under these circumstances, the stomach cannot so readily compensate the obstruction to the propulsion of chyle by the unfavorable position. Decided insufficiency readily takes place. The symptoms of the patient all distinctly point to the fact that the corset is the disturber of the peace, for, after discarding it, decided amelioration is noted. Gradually the gastric catarrh disappears, and, on resuming the corset, the patient readily becomes accustomed to her instrument of torture.

In these cases, therefore, a slight gastroptosis persists which usually cannot be determined by the ordinary clinical methods of examination, unless it be by decided inflation of the stomach. As a rule, symptoms appear only when an error in diet produces an acute gastric catarrh. While the consequences of such a gastric disturbance in patients with a normal stomach soon disappear under suitable treatment, in those with a latent gastroptosis a high-graded insufficiency of the stomach occasionally develops. A loud, splashing sound most distinctly reveals the enlarge-

ment of the stomach. The greater the distention of the stomach in the course of the affection, the more difficult, naturally, is the propulsion of chyle into the duodenum. The affection, therefore, spontaneously forms a vicious circle. An immoderate dilatation of the stomach is also promoted when the belly wall is very flaccid. Even in young girls who are unaccustomed to bodily labor, the muscles exerted in the abdominal press form only a flaccid, thin wall. The activity of the abdominal muscles is still further impaired in women by successive labors, the consequent pendulous abdomen described by Landau [1] becoming particularly noticeable.

The persistence of gastric catarrh or gastric insufficiency in gastroptosis, however, leads to other dangers. Being insufficiently nourished the organism is forced by metabolism to the combustion of its body substance, the fat in the abdomen and the mesentery being first utilized for this purpose. Here we note the well known fact that in an antifat treatment

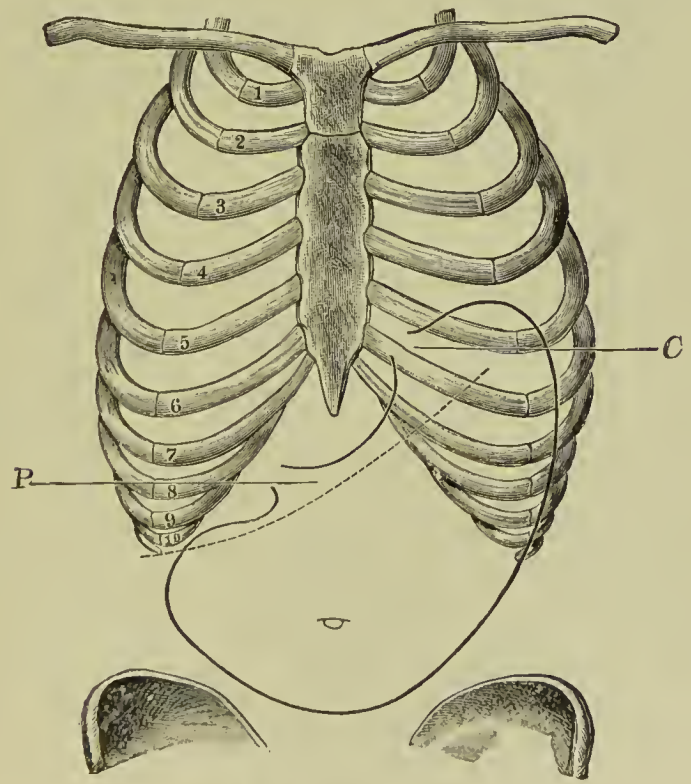

FIG. 14.—Gastroptosis of the third degree.

a decrease in the circumference of the waist is most perceptible. The loss of fat accumulated in the abdominal cavity makes itself felt in the same way as a weakening of the abdominal walls. The position and attachment of the organs are no longer secured by simultaneous pressure, but these depend upon the suspensory ligaments which may readily yield. In

[1] *Landau, Verhandl. der Berl. med. Gesellschaft*, 1890.

severe diseases, such as gall-stone colic, gastric ulcer, or gastric cancer, this dilatation of the prolapsed stomach becomes noticeable much more frequently than after simple gastric catarrh. Its manner of development is interesting, and is as follows: I have repeatedly heard young persons who have recovered from an appendicitis operation state that in the first

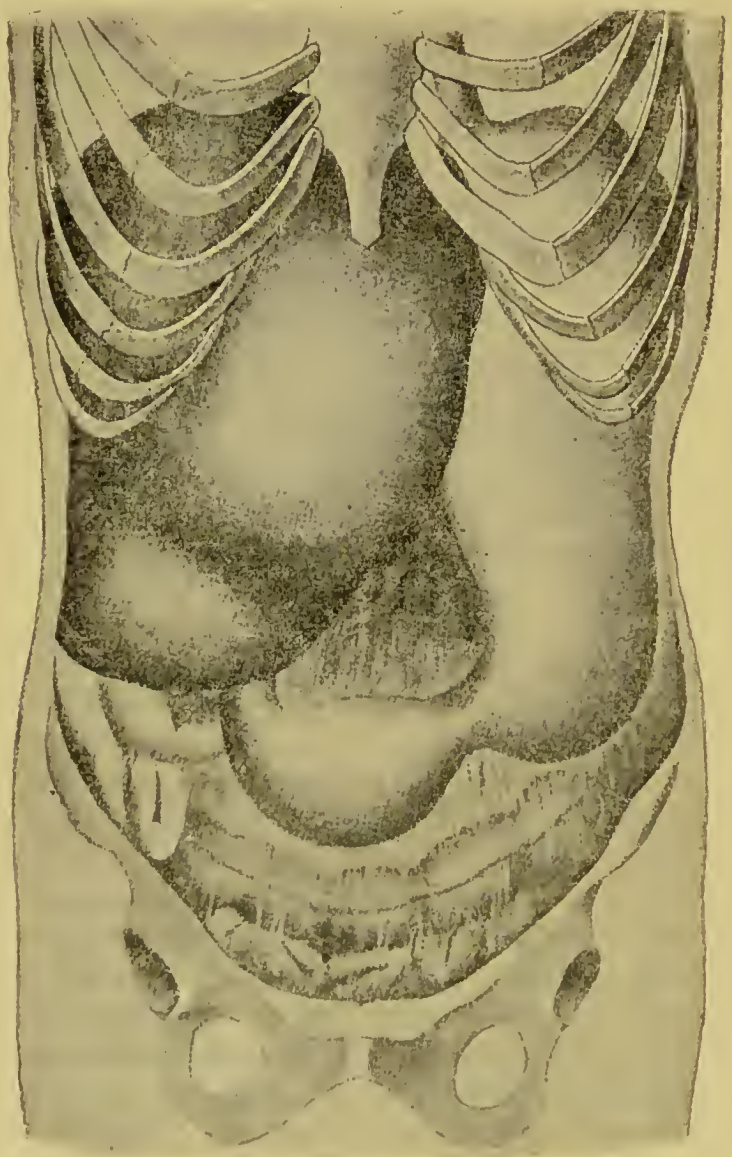

Fig. 15.—Constriction of the right lobe of the liver and of the pylorus.

few days after they had left their beds they observed an increase in the circumference of the abdomen, which was confirmed by the objective examination of the physician. In these cases there was no abdominal hernia, but an enlargement and displacement of the stomach was evident without any symptom of gastric disease. This was explained by the marked disappearance of fatty tissue in the abdominal cavity, since the persons in

question had lost from 11 to 16½ lbs. in weight. A few days after getting out of bed, and being in an upright position, they were well enough to take large quantities of food, and this had caused the over-burdened and distended stomach with all of the other viscera to descend.

That occasionally a decided displacement of the stomach may be determined in kyphoscoliosis is readily comprehended. In these cases, also, the prolapse usually occurs without producing any disturbance in gastric activity (Fleiner [1]).

Lordosis of the vertebral column, which sometimes develops to a decided degree in women predisposed to it if they wear high heels, produces a condition in which the pressure of the viscera dilates the abdominal walls downward and anteriorly, and the internal organs themselves drop (Cséry, Korányi, and Meltzing [2]).

In some diseases, for instance, tuberculosis, the effect of mechanical shock makes itself felt in the abdominal cavity. The paroxysmal shock to the thorax and abdominal wall produced by cough must assist this process, particularly when the factors we have mentioned which favor the descent of the stomach have already been active.

From these descriptions it is obvious that gastroptosis may be looked upon as a disease in only a small number of cases when, from any complication, the compensation is lacking which would otherwise be produced by increased activity of the muscular structure of the pylorus. In regard to the frequency of these disturbances, no absolute general rules can be laid down.

Gastroptosis and enteroptosis are most frequently met with in patients suffering from gastric disease. Thus Einhorn [3] reports that within a few months he found 9 cases among 141 male patients with gastric disease, and among 92 women he found these changes 32 times. It is true that the more vigilantly we search for changes in position, the more frequently we will diagnosticate them in a greater or less degree. Often the appearance of hysteria or neurasthenia leads us to suspect gastroptosis. It may then be doubtful which affection existed first.

SYMPTOMS

Symptoms of gastroptosis may be entirely absent. In young girls who press the pylorus down by wearing a corset, possibly because of the increased difficulty with which the chyle is propelled into the duodenum, eructations will often occur, and the peculiar rumbling or gurgling frequently heard in women who lace very tight is declared by Fleiner to be

[1] *Fleiner, Münch. med. Wochenschr.* 1895, Nr. 42–44.

[2] *Korányi, Berl. kl. Wochenschr.*, 1890, Nr. 31; *Meltzing, Volkmann'sche Vortr.* 1896; *Cséry, Wiener med. Wochenschr.*, 1901, Nr. 28.

[3] *M. Einhorn*, "Die Krankheiten des Magens," Berlin, 1898, page 270.

a murmur of stenosis due to compression of the stomach. In most cases, however, as has been stated, symptoms are either absent, or these mild signs are regarded by healthy persons as due to a disturbance of gastric movement. In weak, anemic persons with a tendency to chlorosis the constriction of the stomach may exert such a deleterious effect that not only the signs of gastric catarrh but also manifold nervous disturbances may present themselves. The symptoms due to gastroptosis are then closely interningled with those of chlorosis, of general debility, and of nervousness, but are, in the main, attributed to chlorosis; they may continue for weeks, months, or even for years. Quite properly Meinert attributes the old axiom, founded upon experience, that marriage is the remedy for chlorosis to the fact that when pregnancy occurs the growing uterus forces the distended stomach upward again.

The symptoms which subsequently arise, provided no intercurrent disease suddenly produce marked insufficiency of gastric activity, as in the manner I have described, resemble those which generally appear in persons suffering from gastric catarrh. A conspicuous symptom in such patients is the great weakness and debility which cannot be accounted for by any preceding error in diet. In the horizontal position, especially in the right lateral, the symptoms ameliorate, for then the propulsion of food goes on under the most favorable mechanical conditions. Vomiting is rarely mentioned in the history, but is most frequent at the onset, and very rare in the later course. Incessant vomiting throughout the entire period of the disease might therefore be utilized as excluding gastroptosis.

The patients almost invariably complain of marked constipation, for which various causes are assigned. In the first place, a displacement of the colon, particularly of the mesocolon in which the thin, fluid feces first become more compact, may cause kinking and, naturally, a retardation of the feces. However, too much stress is not to be laid upon this circumstance, since, after the removal of other causes, and in spite of the persistently abnormal course of the colon, regular fecal discharges almost always take place. It is significant that these women—and with such we usually deal in gastroptosis—generally prefer easily digested food which leaves but little residue in the intestine. We know from physiology that with food consisting chiefly of meat and wheat bread the fecal mass in twenty-four hours amounts to only 100 to 130 grams of moist, and from 20 to 30 grams of dry, substance, while with a diet rich in cellulose, therefore with the ingestion of much rye bread, the feces amount to 400 to 600 grams of moist, and 60 to 80 grams of dry, substance. As the intake of food in gastric affections is generally even less, and accordingly there is less residue, a fecal evacuation every two or three days appears quite natural. Moreover, a pendulous abdomen decidedly decreases the force of the abdominal press, so that small fecal masses formed from easily digested food are only evacuated by great effort. Finally, in my experience, the lack

of muscular exercise on the part of most women adds much to torpidity of the bowels, which therefore cannot be looked upon as entirely the consequence of gastroptosis, coloptosis or enteroptosis.

Among the objective symptoms, a loud splashing sound in the umbilical region is noted by the physician. This sign is so characteristic that when absent in repeated examinations one or two hours after the ingestion of fluid, insufficiency of the displaced stomach may be denied. The importance of the succussion sound in judging of the gastric activity has lately been the subject of much controversy. Although I am willing to admit that this symptom may occasionally be observed in thin, healthy persons, nevertheless I coincide with Stiller and Kuttner[1] in the view that, under these circumstances, the gastric wall is incapable of contracting strongly enough about its contents. In gastroptosis the splashing sound is usually detected around the umbilicus, often, however, below this region.

Among other signs epigastric pulsation, which is emphasized by Glénard, must be considered. It is not of the same diagnostic import as the succussion sound, for, in the first place, when the abdominal walls are thin and the pylorus is situated in the scrobiculus cordis, the pulsation of the vessels may sometimes be felt through the gastric walls, and secondly, when the abdominal walls are rich in adipose tissue, pulsation is unrecognizable notwithstanding the existence of gastroptosis.

Another symptom, a feeling as of the pressure of a girdle in the gastric region, the "corde colique," leaves us completely in doubt. Glénard believes this band to be the colon, while, according to Ewald, the area of resistance, which, however, can rarely be felt, is to be looked upon as the pancreas. This fact is not generally known; it is, however, occasionally of great practical value, for, otherwise, upon palpation in this region, and when there is a disturbance of gastric activity, we might mistake this resistance for a neoplasm. I know of a case in which such an error led to a laparotomy being performed—a state of things naturally not very desirable, yet not so serious as the inverse assumption that a neoplasm is only the head of the pancreas.

The most signal proof of gastroptosis, or that the disturbances present are chiefly due to displacement of the stomach, is furnished us by the movability of other abdominal organs, particularly of the kidneys. In how far it is possible to palpate the kidneys will be discussed later. In almost every marked case of gastroptosis it is possible distinctly to palpate the kidneys, at least the right one, upon the posterior wall of the abdomen. Often one or both kidneys are movable in the abdominal cavity, or they are shown to be fixed in certain areas.

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[1] *Berliner klin. Wochenschr.*, 1901, Nr. 50.

DIAGNOSIS

Naturally the diagnosis is assisted when the walls are very flaccid. In the majority of cases of gastroptosis the distention of the stomach is so marked that the findings appear to be entirely out of proportion to the symptoms, which have usually existed but a short time. In gastrectasis, which develops in consequence of some other gastric affection, derangement of the activity of the stomach has gradually become marked. Finally, we must remember that a displacement may have occurred secondarily from the cicatrix of an ulcer or a neoplasm which had narrowed the stomach, and that dilatation had subsequently occurred. In practice, therefore, the proof of a displacement is naturally much less significant than the recognition of some other obstruction to the propulsion of the ingesta through the pylorus. For this reason, in the examination of a patient suffering from gastric disease, I must advise against a great inflation of the stomach, provided we desire only to determine gastroptosis or the tendency to displacement. *In my experience, only in hysterical, very nervous persons, or in those debilitated from disease of some other organ, will gastroptosis cause a decided disturbance in nutrition.* On the contrary, examination should reveal the cause of the compensatory disturbance in the activity of the displaced stomach. According to the age, the sex, and the general appearance of the patient, we must search for *gastric ulcer,* for *malignant disease of the gastrointestinal canal,* for *cholelithiasis,* or for *pulmonary tuberculosis.*

TREATMENT

In the treatment, prophylaxis plays the first rôle. By prohibiting lacing the chief cause of the development of gastroptosis, as well as displacement of other organs, will be removed. Probably every woman will assure the physician that she does not lace too tight, although an examination of the skin will reveal distinct lines about the waist due to the constricting corset. In regard to these views there is little unanimity between physicians and their women patients. A corset of stiff material worn by those anemic women, weak in muscle, who form the majority of the well-to-do classes, may have the advantage that it gives a certain support to the weak vertebral column which is lacking in strength of muscle and will remedy defects in the carriage of the body. But, as a rule, the physician must insist that the corset be not worn too tight, nor the skirts suspended from the waist. This danger is greatest in girls from fourteen to sixteen years of age whose hips have not yet sufficiently developed. The skirts must be suspended from the shoulders like the trousers of men, and a belt must not be worn. It is well known that the "dress reform," which has already become somewhat general in Germany, is directed toward this purpose. Unfortunately, stout women refuse to

abandon the corset, because without it the waist appears to them to be too large, and the bust is not sufficiently supported. It is to be hoped, although it is by no means certain, that in this respect common sense will finally prevail over vanity and the present standards of beauty.

On account of the tendency of the vertebral column to marked lordosis, the wearing of high-heeled shoes should be prohibited, the height of the heel being limited to 2 or 3 cm.

The formation of a pendulous abdomen in consequence of childbirth may be prevented by maintaining the recumbent posture as long as possible after labor, and on rising from bed the flaccid abdominal walls should be firmly supported by a well-fitting bandage.

The most effectual means to prevent the displacement of organs probably lies in the education of the female from infancy as to the value of physical exercise. With this we might hope that the abdominal, as well as all the other muscles of the body, would attain their fullest development, and that the wearing of sensible clothing by the majority of women would prevent many other disabilities of civilized people.

If a displacement of the stomach has already occurred, yet no symptoms are present, treatment may appear to be superfluous. Nevertheless, in such cases I must advise prophylactic interference and the interdiction of tight lacing, especially if occasional symptoms, such as loud gurgling in the intestines, have already been noted. If there are distinct signs of insufficiency of the stomach, a binder which supports the lower parts of the abdomen is serviceable. An abdominal bandage lately advised by Ostertag[1] is especially valuable for this purpose. In women the symptoms sometimes disappear immediately on its use.

In advising an abdominal binder it must be borne in mind that it would be the height of folly to forbid, on the one hand, the constricting corset, and then, for the opposite effect, to recommend an abdominal bandage which compresses the organs from the waist downward.

According to the statements of Landau and Bardenheuer, there are stays supplied by instrument makers which exert no special pressure about the waist, and to this the abdominal bandage for the lower parts of the abdomen is already attached. I advise these corsets in antifat cures even if there is no gastroptosis, since in hyponutrition, as already remarked, fat disappears from the abdominal cavity with comparative rapidity and, therefore, the condition for displacement of organs is already present. Bial[2] has lately proven by the illumination of the organ that a prolapsed stomach cannot be supported by a bandage. He also relates his experiences, according to which the desired result may be attained by suitable hydrotherapeutic and dietetic measures. Nevertheless I advise

[1] *Ostertag, Monatsschr. f. Geburtsh. u. Gyn., XV.*

[2] *Bial, Verh. d. Congr. f. innere Med.*, 1897, p. 521.

the employment of these abdominal supporters. In the first place, in women the weakened abdominal wall is strengthened, and thereby the danger of a marked displacement of the abdominal organs is averted. Moreover, it is quite clear to me that not only does the motor activity of the stomach improve, but the action of the bowels is decidedly facilitated. The descent of the diaphragm, which by the abdominal press forces the abdominal viscera downward, encounters a stronger resistance in the abdominal bandage than in the flaccid belly walls, which, when strong pressure is exerted, invariably become more distended.

Dietetic rules form an important part of the treatment. From a purely mechanical standpoint the difficulty of propelling the ingesta from the stomach is at once obvious. v. Mering [1] has lately pointed out that in the right lateral position the stomach is most rapidly emptied, and, under the conditions here discussed, we should utilize this fact. In debilitated persons, a rest of one or two hours in the right lateral position after the principal meal should certainly be advised.

Furthermore, it is unwise to permit too copious meals. At least while active symptoms are present, several small meals must take the place of the chief meal. Oertel and Schweninger's advice to the obese not to drink while eating, which they erroneously regarded as an important law, is, on the contrary, well adapted to gastric patients, and particularly to those suffering from gastroptosis.

Under some circumstances we may attempt to hasten the evacuation of the stomach by massage. In most cases I believe this effect is to be attributed to autosuggestion. Nevertheless, I have several times seen such favorable results that I frequently advise it, and the more so since it may be readily practised by the patients upon themselves. The hand rests upon the left arch of the ribs, and about this point rotary motions are made from the left below to the right upward. Massage by another person I do not advise, because, particularly with movable kidneys, there is danger that these may be irritated—a danger which cannot be excluded.

I am, therefore, decidedly opposed to the advice given by some authors to treat gastroptosis by a course of massage of from six to eight weeks, in which the massage is given one or two hours before eating. This will not bring about a true cure of gastroptosis, and a temporary insufficiency of the musculature of the stomach may more certainly be made to disappear by other means.

In the choice of food the same rules are operative which are applicable to all patients suffering from gastric disease. When there is marked disturbance of gastric digestion small quantities of milk may first be permitted, and are very serviceable as easily digested, rich, nutritive products. It is not advisable to give more than one liter of milk in the course of

[1] v. *Mering, Therapie der Gegenwart*, 1902, Heft 5.

the day; more than this will over-burden the stomach. If there is repugnance to milk, cream may be given, more or less fatty according to the severity of the condition, and some tea. Zwiebaek, wheat bread, lean meat, eggs, green vegetables are the foods which next come into question. We must be careful with those foods which readily generate fermentation and gas in the stomach, therefore those especially rich in sugar. Naturally, the individual taste and the experience of special patients must here be taken into consideration. In regulating the bowels we must always consider whether a food shows a strong tendency to generate gas. In constipation we are generally inclined to advise foods rich in cellulose, consequently bread rich in gluten. The cellulose of gluten at first mechanically stimulates the intestine, subsequently it undergoes decomposition in the intestine by the activity of baeteria, and thus volatile acids and gases are formed. In gastroptosis, on account of the pendulous belly which usually coexists, gas production is probably already present and very disagreeable to the patient, so that, under these circumstances, the physician must be cautious. Here fruit, stewed fruit, and acid fruit wine, buttermilk or milk sugar lemonade are frequently preferable to the coarser varieties of rye bread.

As in all displacements of organs we must consider in each individual case whether it is wise to attempt a "fattening cure" by profuse nourishment, so that the fat which has disappeared from the abdominal cavity may again accumulate there. I believe that this question should generally be answered in the affirmative. Patients with an affection of the stomach are usually so debilitated that even a gain in weight of a few pounds is worth our efforts, because this furnishes the best proof of the retardation of the pathologic phenomena. In all cases of hypernutrition prolonged rest is absolutely necessary, for we must never forget that the displaced stomach, even more than the normal stomach, is burdened by the ingested food. The more food, therefore, the patient eats, the less should the stomach labor under unfavorable mechanical conditions. As already stated, it is not enough that the patient assume the lateral position for some time after eating, but for the whole day, uninterruptedly, or at least for the greater part of the day, he should remain in bed or upon a lounge. The too free consumption of milk—over three liters daily, according to the advice of Weir Mitchell—as was formerly and is even now the general rule, can only aggravate the insufficiency of the displaced organ by overburdening the stomach. On the other hand, easily digested fats, such as cream, butter, bacon, or some carbohydrates, such as milk sugar and mannite, should be employed. Alcohol[1] can rarely be dispensed with in the form of brandy, good wine, or even as beer. But only slight quantities

[1] Compare *F. Hirschfeld*, "Die Ueberernährung und die Unterernährung," Frankfurt, 1897, p. 43 u. f.

of the last named stimulant should be allowed, and never more than 200 c.c. at a meal. Brandy may be advantageously given with milk, or even with cream provided it be not too fat. Not only the property of alcohol as a nutrient, but also its stimulating effect upon the gastric activity, is especially desirable (G. Klemperer).

In gastroptosis I have made but little use of drugs to improve the nutrition. Sodium bicarbonate or magnesia, about as much as will cover the tip of a knife, frequently lessens the gastric symptoms. Hydrochloric acid, as a rule, unquestionably has a deleterious effect. A favorable influence is usually obtained by the employment of bitters; and I have frequently used the following prescription:

℞ Tinct. Cinchona comp..... 20.0
Tinct. Nucis Vomicæ..... 5.0
30 drops in water or brandy three or four times daily.

In gastroptosis a mineral spring treatment is naturally not advisable. Even in Carlsbad, particularly in young, nervous persons, such treatment of these cases is often ineffectual. A residence in a place of high altitude is more beneficial, and care must be taken, especially at first, to avoid too great bodily exertion, or to see, at least, that it corresponds with the strength of the patient. In nervous patients it is easily understood that rest in a sanatorium, or a residence with surroundings of beautiful scenery, will have a favorable effect. Sea baths, also, on account of their stimulation of the appetite, are worthy of consideration in some cases, but the physician should always be consulted in these instances. In Germany, bathing in the Baltic Sea is generally more beneficial to such patients than in the North Sea.

Finally, in gastroptosis, as in so many other diseases, hydrotherapy may be employed, and is particularly adapted to the many nervous symptoms which develop in connection with all gastric affections.

Buxbaum [1] and Winternitz advise quick, cool Sitz baths, while others (Matthes, Boas [2]) have seen favorable results from a needle douche which may be used either cold or, like the so-called Scotch douche, cool and warm alternately. In this treatment, other measures may occasionally precede the local douche, as a lukewarm full bath, or general friction of the entire body.

For the sake of completeness I shall only mention that electrical treatment of the stomach, the faradic as well as the galvanic current, is occasionally employed in gastroptosis. I saw favorable results in a case in

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[1] *Buxbaum*, "Festschrift für Winternitz," 1892.

[2] *Matthes*, "Klinische Hydrotherapie," Jena, 1900, p. 226; *Boas*, *Internat. klin. Rundschau*, 1894, Nr. 6.

which almost all other remedies had proven ineffectual. The patient was a lady, exceedingly nervous, who complained of spasmodic pain several hours after taking food.

In the treatment of all cases of gastroptosis the following must be remembered:

We may relieve and improve a state of insufficieney of the gastric musculature which develops in consequence of an unfavorable position of the stomaeh, and brings about severe nutritive disturbance in the patient. The low position of the pylorus, however, remains, and, as is the case with a damaged eardiac valve, only hypertrophy of the cardiac muscle produees compensation; in the same way greater labor is demanded of the musculature of the pylorus on account of its unfavorable effect upon the propulsion of the chyle. The efforts of the physician must be directed, on the one hand, to removing the disturbance of compensation which has oecurred, and he must subsequently try to prevent such a condition. This part of his work is praetical and of the utmost importance. An attempt should also be made to strengthen the abdominal walls and, in so far as possible, to prevent the patient from over-burdening his stomaeh. In how far this may be done in the individual case by giving minute directions in regard to the food can be ascertained only by careful observation.

The surgieal treatment of gastroptosis rarely comes into question, because the eontinuanee of the symptoms, notwithstanding eareful treatment, must probably be attributed to a disturbance of the nervous system. Duret and Rowsing have proposed to attach the gastric wall to the anterior parietal peritoneum. Bier [1] advises us to shorten the gastrohepatic ligament by folding and stitching it, and in four cases he had favorable results by this means. The previous nutritive condition, which was very poor, the cachexia of gastroptosis, and the nervous symptoms were said to be relieved by this treatment.

DISPLACEMENT OF THE KIDNEYS—WANDERING KIDNEY—NEPHROPTOSIS

While displaeement of the stomaeh almost invariably develops after puberty, displaeement of the kidneys is often observed in children. This anomaly was even noted in a nursling.[2]

A male nursling, a few days after birth, was attacked by serious vomiting and symptoms of collapse. Physical examination revealed a small oval tumor at the right arch of the ribs which could be moved over the entire abdomen. The other kidney was also movable, but not to the same extent. The attacks recurred in the next few days, spasms simultaneously taking place. There was marked constipation.

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[1] *Bier, Zeitschr. f. Chirurgie*, 1901.

[2] *Rees Phillips, The Lancet*, 1903, p. 731.

A case of this kind shows that displacement of the kidneys may be congenital. In by far the great majority of cases, however, it develops gradually, as is evident from the following statistics of L. Kuttner.[1]

The age in 326 cases was as follows:

1–10	10–20	20–30	30–40	40–50	50–60	60–70 years.
6	32	82	123	49	26	8

The right kidney is most frequently displaced. According to Ewald and Kuttner, we may assume that the right kidney is seven or eight times more frequently displaced than the left kidney. Often the position of both kidneys is changed, and in the majority of cases greater movability may be determined in the right than in the left. E. Hahn [2] attributes this to the firm attachment of the left kidney to the descending colon (which is little inclined to displacement) and to the pancreas and the spleen, while the right kidney possesses but one point of fastening, the hepatorenal ligament. The sexes are affected by this anomaly in about the proportion of 7 or 8 to 1; among 97 cases of nephroptosis Landau [3] found 10 to occur in men and 87 in women. Hahn, it is true, assumed that floating kidney was 20 times more frequent in women than in men.

In regard to the absolute proportion of cases showing change of position it is difficult to give a positive opinion. From investigations in the living subject, Ewald and Kuttner assume 15 per cent., and with this the reports of anatomists coincide. In the reports of the seventh decade of the preceding century, among the autopsies of the pathological institutes of Berlin, Vienna and Kiel, movability of the kidney was found in only 0.1 : 0.4 per cent., but the more recent investigations of Fischer-Benzon, in Kiel, show from 17 to 22 per cent.

According to the degree of movability of the kidney we differentiate various grades. Under physiologic conditions, the kidneys move slightly from their position with each respiration, descending on inspiration, and rising on expiration. Some clinicians, such as Israel, Litten, Lennhof and Becher, maintain that this movability may be determined by palpation even during life, while the majority of authors deny this. At all events, therefore, in the normal person this movability must be very slight, and in the majority of healthy adults the kidney cannot be palpated from the abdomen. In the minority this is to some extent possible, but only the lower third of the organ can be felt. Of course, thin, flaccid abdominal

[1] *L. Kuttner, Berl. klin. Wochenschr.*, 1890, p. 364; compare in the same journal Ewald's lecture before the Berlin Medical Society, March, 1890, and the discussion in connection with it. (Virchow, Litten, Landau, P. Guttmann, Senator, Israel and others.)

[2] *E. Hahn, Zeitschr. f. Chirurgie*, LXVII, p. 356 *et seq.*

[3] *L. Landau*, "Die Wanderniere," Berlin, 1881.

walls will extraordinarily facilitate the examination. Becher and Lennhof [1] point to a special structure of the body which lends itself to palpation. Although tall persons usually form the bulk of this group, a definite bodily proportion is decisive, and this may be found in individual persons and in various races under different conditions. The greater the distance from the manubrium sterni to the symphysis pubis, the greater the portion of the kidney susceptible to palpation. If the slight physiologic movability of the kidney is increased, it is possible during palpation of the abdomen not only to feel a small portion of the kidney but two-thirds, or even the complete extent, of the organ; under the influence of the respiratory movements the displacement becomes particularly prominent. In a later stage of abnormal movability, the kidney can no longer be recognized in its normal position upon the posterior wall of the abdominal cavity, but may be felt in some other area of the abdomen, and thence it may either be readily moved or it becomes adherent to this region so that it can only be displaced in connection with other abdominal viscera.

ETIOLOGY

A primal cause of abnormal movability is probably a congenital predisposition. In the normal person the kidneys are usually situated in a groove in the posterior abdominal wall, anterior to and alongside the transverse processes of the vertebræ, and extending from about the height of the twelfth cervical to the third lumbar vertebra. Two Russian investigators, Wolkow and Delitzin,[2] have especially called attention to the importance of the formation of this paravertebral niche for holding the kidney. If this niche is markedly flattened, the kidney, naturally, will much more readily slip out. Besides this congenital flatness of the groove, which is readily seen, scoliosis of the vertebral column adds to the condition. We must also consider that in intra-uterine life the kidneys are low down in the abdominal cavity in the sacral hollow of the pelvis. Gradually they move up higher. This shows at once that the kidneys are not supplied with very tense suspensory ligaments; they are partially held by their ligamentous apparatus, but are also supported by the peritoneal layers and the fatty capsule; on the other hand, the kidneys maintain their position by the pressure of all the abdominal viscera. Accordingly, by a sundering of various links, this girdle which supports them may become slack or broken. A flattening of the niche simultaneously with a loosening of the ligamentous apparatus seldom causes a displacement of the kidney. Rigid abdominal walls, such as are found in most young,

[1] *W. Becher* und *R. Lennhof, Deutsche med. Wochenschr.*, 1898, Nr. 32, und *Verh. d. XVIII. Congr. f. inn. Med.*, p. 476.

[2] *Wolkow* und *Delitzin*, "Die Wanderniere," Berlin, 1899; compare also *M. Zondek*, "Die Topographie der Niere," Berlin, 1903.

robust persons, usually hold the abdominal organs in their natural position. This explains the rarity of floating kidney in persons under twenty years of age. When, from any circumstance, pressure in the abdomen is lessened, this favorable influence is lacking. Lacing is the most frequent among the deleterious factors which must here be considered, because the liver is thereby pressed downward, and the right kidney, which is intimately connected with the liver, also has pressure exerted upon it. This explains the fact that the right kidney shows abnormal displacement so much more frequently than the left. We, however, are under the impression that lacing is here not so predisposing as is the case in gastroptosis. Repeated pregnancies which markedly diminish the support of the abdominal wall mostly have a decided effect in that a portion of the abdominal contents, usually the intestines, bulge forward and downward, so that lateral pressure upon the kidneys is diminished.

The greatest decrease in pressure occurs when the abdominal cavity suddenly becomes smaller, as after the operative removal of large tumors. Under such circumstances, Israel noted that, before his very eyes, the kidneys became abnormally movable.

A gradual decrease in pressure may also be expected when from general emaciation considerable adipose tissue disappears from the abdominal cavity. Other deleterious effects may also be operative, such as shock to the abdominal wall. Thus I would explain the wandering kidney which I several times saw develop in emaciated patients in the course of pulmonary disease. In emphysema and asthma floating kidney may easily be produced, because, from the emphysematously distended lung, pressure is exerted upon the diaphragm, and then referred to the liver and the right kidney. Repeated shock to the abdominal wall from paroxysms of cough may gradually loosen the kidneys. In one case of wandering kidney causing abdominal symptoms the patient attributed it to prolonged laughter. In a similar manner straining during a difficult fecal evacuation, or in lifting a heavy load, may be the cause. Under such circumstances traumatic origin of wandering kidney is quite possible, while in a normal, well-fastened kidney this theory is scarcely plausible.

Just as the stomach may subsequently be displaced by a neoplasm which adds to its weight and dilates it, so may this condition result in the kidney from hydronephrosis or pyonephrosis. Such cases, of course, are not wandering kidneys in the usual acceptation of the term.

SYMPTOMS

Symptoms of wandering kidney may be entirely absent. The patients, in this instance, are usually strong, plethoric persons otherwise quite well, whose kidneys, one or both, have descended into the abdomen without causing any symptoms. Occasionally abnormal sensations are noted which

incidentally make known to the person in question that he has a movable kidney. Some patients report after unusual exertion a sensation of rumbling or one like the gliding about of a heavy substance. It is difficult to decide whether these reports depend merely upon auto-suggestion, or to what extent indefinable sensations, due to the influence of already existent factors, may subsequently be explained. It is still more difficult to assign vague nervous symptoms, whatever their nature, to nephroptosis. While we cannot doubt that all varieties of nervous symptoms may appear in persons whose kidneys are normally attached, yet, when people are predisposed to nervous affections, we are justified in assuming a movable kidney to be the cause of various painful sensations such as sciatica, intercostal neuralgia, or similar affections. Sensitiveness in the region of the iliac fossa may be attributed to the kidney. In any case the physician is wise not at once to communicate the fact that during an examination he has accidentally discovered a wandering kidney. The precautions yet to be considered, such as wearing a bandage, refraining from lacing, etc., can also be insisted upon when we speak to the patient merely of "*a predisposition to wandering kidney,*" and the first duty is to prevent the production of a floating kidney.

Symptoms on the part of the stomach are most frequently noticed, and these usually simulate a chronic gastric catarrh; acute attacks, designated as gastric spasm, are very rare. As a rule, we note loss of appetite, eructations, and pressure in the gastric region, less frequently vomiting. We know from numerous observations that in diseases of other organs, and particularly in those of the abdomen, the stomach is likely to become implicated. A heavily coated tongue is almost invariably present in disease of the gall-bladder, of the urinary bladder, and in inflammation of the renal pelvis, and the appetite is always decreased. It must be borne in mind that nephroptosis is often combined with gastroptosis. These patients are usually pale and weak young persons in whom gastric difficulties are prominent; stout, elderly women, even when a pendulous abdomen has developed, are more rarely affected. According to statistics, the implication of the stomach in nephroptosis is about as follows:

In 89 cases of movable kidney Kuttner found the position of the stomach normal in only 10, that is, the greater curvature of the stomach was situated normally in the median line between the umbilicus and the ensiform process.

In 79 cases the greater curvature was 3 to 4 cm. below the navel, occasionally even lower than this; in 15 of these cases, by inflation of the abdomen with air by means of a double bellows, a true prolapse of the stomach could be observed.

Dyspeptic symptoms were present in 70 of these persons, hence this symptom may be designated as the most important and most common in floating kidney.

A peculiarity of the gastric symptoms in nephroptosis is their increase after muscular exertion and their disappearance, or at least improvement, after prolonged rest. As a rule, the majority of these female patients suffer most during the menstrual period.

In nephroptosis, just as in gastroptosis, constipation is important and is due to the same causes—weakness of the abdominal walls, insufficient nourishment or food poor in residue, often kinking of the colon, etc.

Symptoms on the part of the kidneys are rare. It might readily be supposed, and would seem likely, that torsion and kinking of the ureters would occur frequently. As a matter of fact such an accident is very rare. Dittl, Landau, Lindner and others have, it is true, described attacks which resembled renal colic and which were mistaken for this. Occasionally it was believed that spasmodic contraction of the ureter could be felt through the abdominal walls. Some patients are attacked so suddenly with sensations of nausea, vomiting, and severe pain in the renal region, the ureters and the bladder, that the physician must assume severe peritoneal irritation, for the pulse also is small and increased, the skin is cool, cold sweat appears, etc. But such intermediate attacks which probably depend upon kinking of the ureter or of the vessels, or torsion of the renal nerves, are rare; I have only once seen a case of the kind. Albuminuria and hematuria are infrequent, as are also so-called intermittent hydronephrosis, pyonephrosis, and pyelitis. Up to the present time but little attention has been given to the latter complication, therefore I shall briefly relate my own experience in regard to it. [These attacks are the so-called "Dittl's crises."]

Pyelitis as a Complication of Hydronephrosis.—The patients, usually women about forty years of age, who for ten or even twenty years have had no symptoms of their wandering kidney, are suddenly attacked with acute pains in the abdomen. The pains, as shown by palpation of the abdomen, are chiefly located in the displaced kidney, and radiate along the course of the ureter to the region of the bladder. Strangury frequently occurs. There is fever from 100.4° to 102.2° F. which is remittent in type. The urine is turbid, usually of acid reaction, and microscopically is composed almost exclusively of pus corpuscles. Chemical examination, of course, reveals albumin, but decided amounts (up to 5 per 1,000) are also found in the filtered urine or in the clear urine after sedimentation has occurred.

After one or two weeks the inflammatory symptoms subside. The temperature becomes normal, but the pulse for some time continues to be accelerated. The sensitiveness of the kidney gradually passes away. The albumin decreases so rapidly that after two or three weeks none can be detected in a clear layer of urine; but the excretion of pus and the turbidity of the urine due to this may continue for months and, occasionally, even for years. For a long time the patients are able to take but

little exercise and must avoid all exertion. Prolonged sitting is impossible. A slight tenderness of the urinary organs upon pressure continues to exist for some time, and makes the wearing of bandages or corsets irksome. *Slow recovery from pyelitis and floating kidney is characteristic.*

In the treatment warm drinks, warm baths, hot poultices, etc., are most effective; among drugs salol, urotropin, uva ursi and the like.

In one case, eight days after a very movable right kidney had become affected I saw the left kidney also attacked, this, so far as could be ascertained, being in its normal situation. The fever, which had declined, again rose, and all of the symptoms returned.

The sudden appearance of pyelitis without other symptoms may be attributed to an acute infection; but the nature of the infection is not clear. If gonorrhea or cystitis have not previously been present, it is very likely due to an emigration of bacteria from the intestine (Posner). But such complications are rare, for Kuttner saw only 4 among his 89 cases, and among 93 cases operated upon by Küster [1] only two displaced kidneys showed the development of pathologic processes.

DIAGNOSIS

The diagnosis of floating kidney is in some cases very easy. Even upon superficial palpation of the abdomen, we feel an organ of the size and consistence of the kidney which either moves about or is fixed in some area. In the majority of cases the organ is situated upon the right side of the body, which confirms the opinion that the right kidney is by far the most frequently displaced. The diagnosis becomes difficult when the kidneys still retain their position in the posterior abdominal wall. Bimanual examination then becomes necessary. As already stated, only in those persons who are tall and thin is it possible to feel a portion of the normally situated kidney, that is, about one-third of the organ, and this is most readily perceptible in the deeper situated right kidney. To overcome the marked tension of the abdominal walls which is often present, Becher and Leunhof advise an occasional examination in a warm bath. A simpler method which I found serviceable was the employment of a moist, hot poultice about the abdomen.[2]

For the examination, the patient is placed in a horizontal position, the legs are flexed somewhat, and abducted and rotated outwardly to ensure a comfortable position, then one hand is placed upon the back at the

[1] *P. Geis*, "18 Jahre Nierenchirurgie," Marburg, 1900.

[2] The relaxation of the tension of the abdominal walls is naturally also of value in the consideration of other pathologic processes. For diagnostic purposes in perityphlitis, the occasional employment of the hydropathic poultice to determine the presence and extension of the exudate is especially useful. The enlarged appendix can then occasionally be palpated.

height of the eleventh or twelfth rib while the other gradually makes deep pressure from above downward. An accumulation of fat, as well as marked tension of the abdominal walls, renders this examination difficult, and even with abundant practice it often gives a doubtful result. If upon respiration the kidney seems to be movable we may draw the conclusion that it has descended somewhat, since, under normal circumstances, this movability is hardly perceptible. Another method of examination, percussion, often leaves us absolutely in the dark.

Renal Dystopia.—In considering the findings, we must first answer the question: Is not the displacement a congenital change, a so-called renal dystopia? Müllerheim [1] has recently taught us by his special investigations that this renal dystopia depends upon an embryonal inhibitive formation, and is by no means rare, for he succeeded in finding 200 cases reported in literature. The characteristics of renal dystopia are, as a rule, the change in form, the fixation of the position, the abnormal vascular supply, and the shortness of the ureters. Some of these characteristics, such as the abnormal vascular supply, cannot be recognized during the life of the patient. More significance is to be attached to the change in form detected by palpation. Frequently the kidneys still show fetal lobulation, or they have coalesced and formed the so-called horseshoe kidney. Sometimes, the dystopic kidney lies in the pelvis. If, however, the assumption of a congenitally displaced kidney is already likely from the fixation, the proof may be found, according to Müllerheim, by measuring the ureters. Normally, these have a length of about 25 cm., but in a dystopic kidney there is often a difference of 10 cm. Unfortunately to take these measures is a very complicated procedure; it can only be done by catheterization of the ureters, and, with a tortuous ureter, this is often deceptive.

The diagnosis "floating kidney" naturally receives some support from the appearance of the person in question. In a patient with a tendency to anemia there must also be either a pendulous abdomen or a somewhat flaccid abdominal wall. A certain degree of "nervousness" or hysteria is significant of this affection. The presence of Stiller's sign, abnormal movability or deficient chondrification of the tenth rib, gives a view of this kind an objective basis. The presence of kyphoscoliosis strengthens the diagnosis of wandering kidney. Naturally, we must remember that many women show a moderate degree of kyphoscoliosis. In men, and in a left-sided floating kidney, the greatest care in the diagnosis is presupposed. The fact that in floating kidney the stomach rarely is normal in position and size may also be utilized in the diagnosis. Finally, an examination of the patient in various positions of the body (upon the side

[1] *R. Müllerheim, Verhandl. der Medicin. Gesellschaft in Berlin*, November, 1892; see also its ensuing discussion.

and upon the back) must be made, the alteration in position of the movable organ must be followed and investigated, and we must observe whether it is connected with other organs. According to J. Israel,[1] the proof that the kidneys are out of their normal positions is alone decisive, since constriction of the right lobe of the liver,[2] carcinomatous and tubercular tumors of the ascending colon and of the flexure of the colon, carcinomata of the pylorus, small ovarian tumors with long pedicles, and enlarged gall-bladder have been mistaken for floating kidney.

(For confusion with wandering spleen see page 293.)

TREATMENT

The treatment of floating kidney must be limited in most cases to the application of a suitable bandage, to preventing the formation of a pendulous belly, and, in case this is already present, preventing its further enlargement. It is easy to understand that the symptoms of wandering kidney are not so readily removed by a bandage as those of gastroptosis. If pyelitis has preceded, such a bandage is often intolerable to the patient.

Occasionally a corset specially designed is useful, yet often this fails to have any effect. Then we are compelled to resort to strips of adhesive plaster. Rose[3] advises the following:

"Three strips as wide as the hand and of varying length are used, the longest being applied firmly around the belly; the entire abdominal mass is firmly pressed upward from below, and the two ends of the adhesive strip are brought together at or over the vertebral column. The crest of the ilium should remain free, but the plaster must be applied immediately above, and adjacent to, this bone. The propping of the abdominal wall is strengthened by the application of two additional lateral strips which also meet at the vertebral column, running upward and backward from Poupart's ligament."

If these bandages do not at once relieve the discomfort of the patient, we should insist upon a few weeks of rest in bed.

Plentiful nourishment and prolonged bodily rest will, as a rule, remove the symptoms which appear in very nervous patients who suffer from floating kidney. By this treatment fat will perhaps be deposited in the belly and the capsules of the kidneys, and thus a cause of abnormal movability will be removed. The nervous symptoms, too, are usually relieved by a rest cure in suitable environment and by the employment of hydrotherapeutic measures. But, in the end, the results do not entirely

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[1] *J. Israel*, "Chirurgische Klinik der Nierenkrankheiten." Berlin, 1901, p. 20.

[2] *Penzoldt, Münchener med. Wochenschr.*, 1903, Nr. 10.

[3] *Rose, Zeitschr. f. prakt. Aerzte*, Sept., 1901; quoted from *B. Presch*, "Die physikalisch-diätetische Therapie in der ärztlichen Praxis." Würzburg, 1903, p. 170.

correspond with our hopes. After a short time, the old complaints of a wandering kidney are again heard.

The inefficacy of treatment indicates that operation for the removal of the symptoms should be considered. The excision of the kidney has been proposed and carried out, but, fortunately, this is no longer advised by any surgeon. The knowledge that when one kidney is very movable the other organ often shows the same disability, although to a less extent, should have prevented such a method of procedure. Even when the operation of suture, yet to be touched upon, would be useless, extirpation must be rejected, for, no matter how great the nervous symptoms, the most important disease is in the nervous system, so that, as a rule, the removal of the kidney brings no permanent improvement. The dangers of this operation may best be seen from E. Hahn's [1] report of 42 extirpations for wandering kidney, with the result that 11 patients succumbed to sepsis, peritonitis and uremia.

The operation usually employed at the present time is suture of the kidney to the abdominal wall (nephrorrhaphy), introduced by E. Hahn. The kidney is exposed by excision in the lumbar region, the fatty capsule is partly removed and fastened to the muscles by a few stitches through the capsula propria and the substance of the kidney. According to Israel, under some circumstances, the repetition of nephrorrhaphy, perhaps by another method, may be necessary. Objections have been raised to this operation. In the first place, Hahn himself emphasizes that it is not quite devoid of danger. The mortality is said to be from 2 to 3 per cent. Furthermore, the cure is often not permanent, the difficulties being, in fact, not alone due to the kidneys, but also to a simultaneously present gastroptosis, and symptoms referable to the stomach and to the nervous system are combined. According to the reports of various surgeons, the cures may be estimated at 50 to 60 per cent. From this the conclusion is obvious that the operation is advisable only in such cases of floating kidney as present also renal symptoms, therefore, symptoms of constriction, of hydronephrosis or pyonephrosis, or a stubborn, distressing pyelitis. This, as has been stated, is only the case in a minority of the patients.

DISPLACEMENT OF THE LIVER—WANDERING LIVER (HEPATOPTOSIS)

Displacement of the liver is comparatively rare, provided we mean the complete descent of this organ from its usual position. Slight degrees of displacement, however, are comparatively frequent, as has been shown when the abdominal cavity has been opened during life. Kehr, therefore, maintains that a somewhat movable liver is found in all women

[1] *E. Hahn, Zeitschr. f. Chirurgie*, LXVII, p. 363.

who have borne children. When the liver descends into the lower abdominal cavity, we naturally first attribute it to an abnormal weakness of the suspensory ligaments, and, secondly, repeated pregnancies which weaken the abdominal walls are frequently held responsible. Thus Landau [1] found a very marked displacement of the liver into the lower abdominal cavity in a woman, aged 28, who in three years had passed through four pregnancies.

Cantani [2] was the first to describe such a change of position, and other cases were subsequently reported by various authors. In 1885, Landau published a comprehensive monograph, "Ueber Wanderleber und Hängebauch," in which these alterations in the position of the liver are accurately described as well as their mechanical effect upon the position of the abdominal organs.

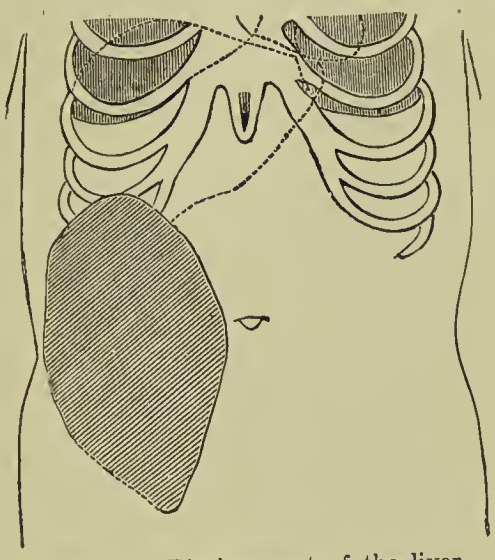

FIG. 16.—Displacement of the liver.

Among 31 cases of floating liver which Landau collected from literature, 27 occurred in women and 4 in men; 14 of these cases were his own patients, and he expressly points out that most authors underestimate the frequency of wandering liver because the liver is rarely examined while the patient is in the erect posture. The age of the person affected is usually over forty years.

Hepatoptosis is recognized by the absence of liver dulness in the usual area, and by the discovery of a movable tumor of the size and consistence of the liver in the lower abdominal cavity; movability generally occurs when the body changes its position. In many cases, displacement of other organs can also be determined; Landau, for instance, among his 14 cases of wandering liver found a floating kidney in 4.

The position of the liver is best shown by the accompanying illustrations taken from Landau's book.

Confusion with cancer of the stomach, tumors of the omentum, and ovarian cysts may occur if we do not bear in mind displacement of the liver, and thus fail to trace the boundaries of the liver in the right hypochondrium.

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[1] *L. Landau*, "Die Wanderleber und der Hängebauch der Frauen," Berlin, 1885, comprehensive compilation of the literature.

[2] *Cantani, Schmidt'sche Jahrbücher*, CXLI.

SYMPTOMS

Symptoms of wandering liver are usually manifest, for the organ is so large that, when in an unusual position, its pressure upon other organs could not be unnoticed. A sensation of fulness, rumbling in the abdomen, ill-defined pains which sometimes extend over the entire abdomen, to the chest, to the small of the back, and to the right shoulder are, according to Landau, the most frequent signs. As in displacement of other organs, the patients complain most after exertion; on prolonged rest in the recumbent posture the symptoms disappear. Some patients are annoyed by the pulsation of the abdominal aorta and other large abdominal vessels which is distinctly felt. Sometimes it is impossible to trace an intimate connection between the nervous symptoms and the abdomen. Disturbances of gastric digestion and of bowel action are often noted, as in displacement of all other abdominal organs.

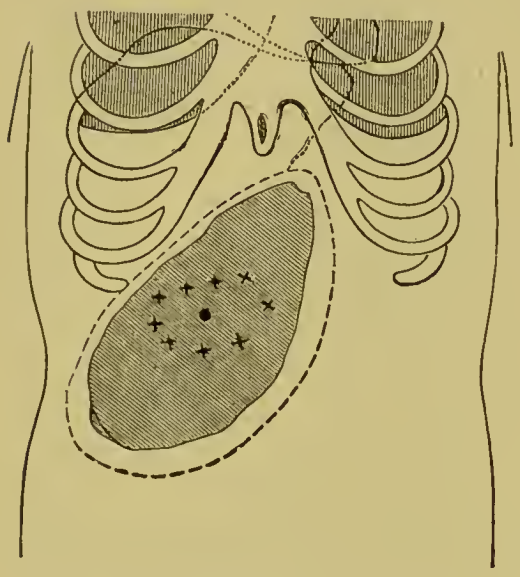

Fig. 17.—Displacement of the liver.

TREATMENT

The treatment generally consists in applying a binder to support the pendulous abdomen. Schott[1] has constructed a shield resembling the human hand to hold the liver in its normal position, but, like all other apparatus to support the abdomen, the results are not very satisfactory.

DISPLACEMENT OF THE SPLEEN—WANDERING SPLEEN

A displacement of the unenlarged, normal spleen is very rare. Glénard reports that he found it only twice in 160 cases of enteroptosis. The location of the spleen near the diaphragm is such that in all pathologic processes which dilate the left thoracic cavity it is forced downward, and thereby is more readily reached by the palpating finger; but there is no greater permanent movability than in the normal organ. A displacement of the spleen is also observed when it becomes enlarged by pathologic processes (by uncompensated valvular lesions, hepatic cirrhosis, malaria, leukemia). But, even in this case, the movability is usually not great. Finally, those cases remain in which extreme movability of the spleen is

[1] *Schott*, "D. Medicinalzeitg." 1882, Nr. 21 und 22.

noted in some area of the abdominal cavity, most frequently in the left iliac fossa. The cause of this displacement is unknown. Flaccidity of the abdominal walls or shock naturally favors its occurrence; but only in a very slight number of persons, and these mostly women, are the ligaments of the spleen so feebly developed as to permit a marked displacement.

DIAGNOSIS

In the diagnosis, a differentiation must be made between wandering spleen and the far more frequent floating kidney. The spleen frequently shows upon its anterior border one or more incisures which are lacking in the kidney. The entire configuration of the spleen is different from that of the kidney. It must be remembered, too, that the right kidney is most likely to be displaced, and this is generally detected, although not always, upon the right side of the abdomen. When the spleen is absent from its normal area on percussion the diagnosis is certain, but this is a proof naturally very difficult to obtain in the obese and in very old persons. The kidneys should either be palpable in their normal position, or their absence decided upon. In conclusion, they may be confounded with very movable ovarian cysts having long pedicles.

SYMPTOMS

Symptoms of wandering spleen may be entirely absent; occasionally there are pains of very indefinite nature. The turning of the pedicle and consequent compression of the splenic vessels may cause atrophy or even gangrene in the surroundings of the organ or in the stomach. In one case the spleen steadily decreases in size, the disturbances gradually disappear, and recovery takes place; in another, peritoneal processes may develop. But these are probably great rarities, always current in medical literature.[1]

TREATMENT

In the treatment, the employment of an abdominal bandage must first be considered; then, by the administration of arsenic, quinin, and iodin, we must attempt to decrease the size of the spleen; finally, the nervous symptoms should be combated by hydrotherapy.

Among surgical measures, fixation of the spleen, splenopexy, which was first proposed by Rydygier[2] is prominent. Litten[3] quite properly

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[1] It is quite remarkable how few reports there are in the literature of the last few decades regarding wandering spleen, which is a proof of the rarity of the affection, for the obverse conclusion that everything relating to this condition has been ascertained, and that, therefore, further reports would be superfluous, is unjustifiable.

[2] *Rydygier, Verhandl. der deutschen Gesellschaft f. Chirurgie*, Berlin, 1895.

[3] *Litten*, "Die Krankheiten der Milz," Wien, 1898, p. 37.

raises to this the objection that permanent fastening of an organ which to so great an extent takes part in the respiratory excursus of the diaphragm can hardly be expected. When the spleen is decidedly enlarged, however, its total removal may be considered. It is self-evident that this is by no means a simple operation [1] and that it should never be advised when nervous symptoms only are present.

DISPLACEMENT OF THE COLON (COLOPTOSIS)

In 1853 Virchow [2] called attention to the fact that displacement of the colon was of frequent occurrence, and attributed this to local chronic peritonitis and to acute, infectious, intestinal diseases, especially dysentery. Displacement of the colon is due to the same causes as general splanchnoptosis, to a weakness of the ligamentous apparatus and a decidedly pendulous abdomen. The greatest deviation in course is shown by the transverse colon, particularly by the right flexure, which occasionally forms a sharp angle whose vertex is situated below the umbilicus, the entire large intestine resembling in shape the letter M. Under these circumstances, particularly in aged persons, portions of the intestine packed with feces are felt in the right iliac fossa, a finding which led me to regard perityphlitis as the consequence of fecal stasis, whereas we now know with certainty that the feces above the ileo-cecal valve are still fluid in composition, the solidification chiefly taking place in the transverse colon.

Displacement of the colon may be proven by inflation from the rectum. Such a process, however, is usually superfluous, because valueless in treatment. For although displacement of the colon may hinder the propulsion of the intestinal contents, as has been stated, an obstruction in the intestinal canal is easily compensated for by a greater activity producing hypertrophy of the smooth musculature of the intestine. The causes of constipation may be readily found in weakness of the abdominal muscles, in the composition of the food which leaves too little residue, and often also in gastric disturbances. Nervous symptoms may usually be attributed to a general splanchnoptosis and to neurasthenia rather than to coloptosis.[3]

The most effective treatment in displacement of the colon as well as any other part of the intestines consists in the application of a well-fitting abdominal binder, and the regulation of the diet and the entire mode of life, in the manner already described.

Just as in gastroptosis, Bier attempted the surgical treatment of colop-

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[1] *Compare Vulpius, Beiträge zur klin. Chirurgie*, XI, 1894; *Kirchhoff, Therapeut. Monatsh.*, 1898.

[2] *Virchow's Archiv*, V, 1853.

[3] *Blocher, Zeitschr. f. Chirurgie*, LVI.

tosis. In a patient who suffered from constipation accompanied by pain in the left side of the abdomen, the mesocolon of the transverse colon, which was extremely tortuous, was straightened by silk sutures.

From the description just given, which shows that the symptoms depend only in small part upon displacement of the colon, it is evident that such surgical treatment is not advisable.

DISPLACEMENT OF THE HEART—WANDERING HEART COR MOBILE [1]

Under physiologic conditions the heart is held in position by the aorta and the pulmonary artery which are firmly adherent to the pericardium, and also by its attachment to the diaphragm in the thorax; it is still further supported by the large veins which empty into the heart. As is shown by the illustration (Fig. 18), taken from Determann's [2] work, the heart is attached at such points on its right and upper side as will permit to the apex of the heart the greatest possible movability in conformity with the movements of the thorax, and this position is most distinctly revealed by the apex beat. As is the case with the abdominal organs, the heart is also maintained in position by the pressure of the adjacent organs. Thus the lungs completely surround the heart and exert pressure which, in the case of pleural effusion or any other process which decreases the space in the thorax, may increase to such an extent as to force the heart away from its normal position. The heart is also held in place by its attachment to the diaphragm, and particularly to the central tendon, by means of which every variation in pressure within the abdominal cavity is transmitted to the heart (see Fig. 19, taken from C. Toldt's [3] *Anatomical Atlas*).

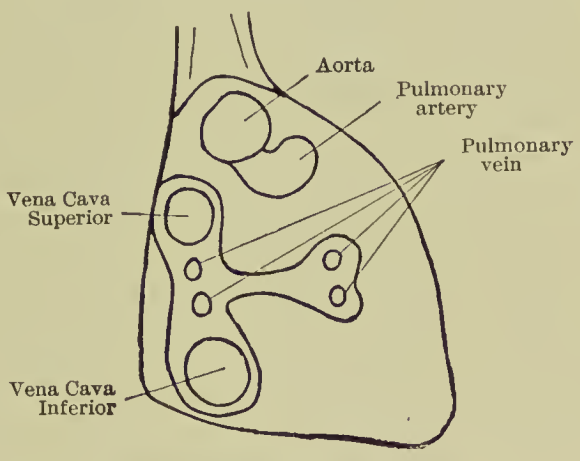

FIG. 18.—Pericardium with vessels. (After Henke.)

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[1] The term employed by some authors, *cardioptosis*, does not appear to me to be happily chosen, in which opinion A. Hoffmann coincides, since it is too suggestive of a low position of the cardia, therefore of a portion of the stomach.

[2] *Determann, Deutsche med. Wochenschr.*, 1900, Nr. 5; compare also the discussion following this.

[3] *C. Toldt, Anatom. Atlas*, III, Aufl., IV, Liefer., p. 482, Berlin und Wien., 1903.

To a certain degree every heart is movable; this has been known to clinicians ever since the introduction of percussion. It has also been known that the heart could be displaced 1 or 2 cm. to the left, and, to a somewhat slighter extent, also to the right. But the investigations of Rumpff, Determann, A. Hoffmann, Cherchewski,[1] L. Braun, Leusser and others have given us more accurate information. Determann, in particular, deserves great credit for having widened our knowledge of this process by a large number of researches, in which the results of percussion were subjected to a subsequent test by X-rays. These results, later confirmed by other investigations, are as follows:

In a normal person in the left lateral position displacement of the heart takes place, upon the average 2 to 3 cm. to the left, and in the right lateral position about 1 cm. A displacement is rarely absent, but occasionally it may be very slight. In some healthy persons, however, this displacement is decided, and amounts to 6 cm. to the left and 4 cm. to the right. Generally, these extreme degrees of displacement are found in middle-aged persons who are poorly nourished and have a weak muscular system, in whom, therefore, an abnormally great displacement of the abdominal organs is also most frequently found. This coincides with the opinion that in women the heart is generally much more movable than in men. In the newborn, cardiac displacement is almost unknown, and in children it is very slight. A deviation from the normal state of the abdominal viscera is found when, in the aged, the movability of the heart does not increase but decreases, probably because, by augmentation of the emphysema, the pressure exerted by the lungs upon the heart becomes greater. But the pressure from the abdominal cavity influences the movability of the heart still more than pressure from the lungs. During pregnancy the heart is forced upward, and rests upon the diaphragm in such a way that hypertrophy is simulated; yet the heart is but slightly movable. Soon after labor, however, the highest grades of cardiac displacement may be noted, as a rule, to the left and upward, so that the apex beat appears near the axillary cavity. A similar influence is exerted by all other processes in the abdomen, such as ascites, meteorism, by a pendulous belly, or enteroptosis.

Emaciation, as a rule, intensifies the movability. In the first place, the layers of fat normally surrounding the heart and which reach their greatest development (Gerhardt) in the obese occasionally disappear; the

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[1] *Cherchewski, Gaz. méd. de Paris*, 1887, No. 53; *Rumpff, Verhandl. d. VI. Congresses f. innere Med.*, 1888; *Deutsche med. Wochenschr.*, 1902, Nr. 31, und 1903, Nr. 3; *A. Hoffmann*, also, 1900, Nr. 19; *L. Braun*, "Ueber Herzbewegung und Herzstoss," Jena, 1898 u. *Centralbl. f. innere Med.*, 1902, Nr. 35; *Leusser, Münchener med. Wochenschrift*, 1902, Nr. 26; *Pick, Wiener klin. Wochenschr.*, 1889, p. 747; *A. Schmidt, Deutsche med. Wochenschr.*, 1901, Nr. 16. I have also made investigations in a small number of cases.

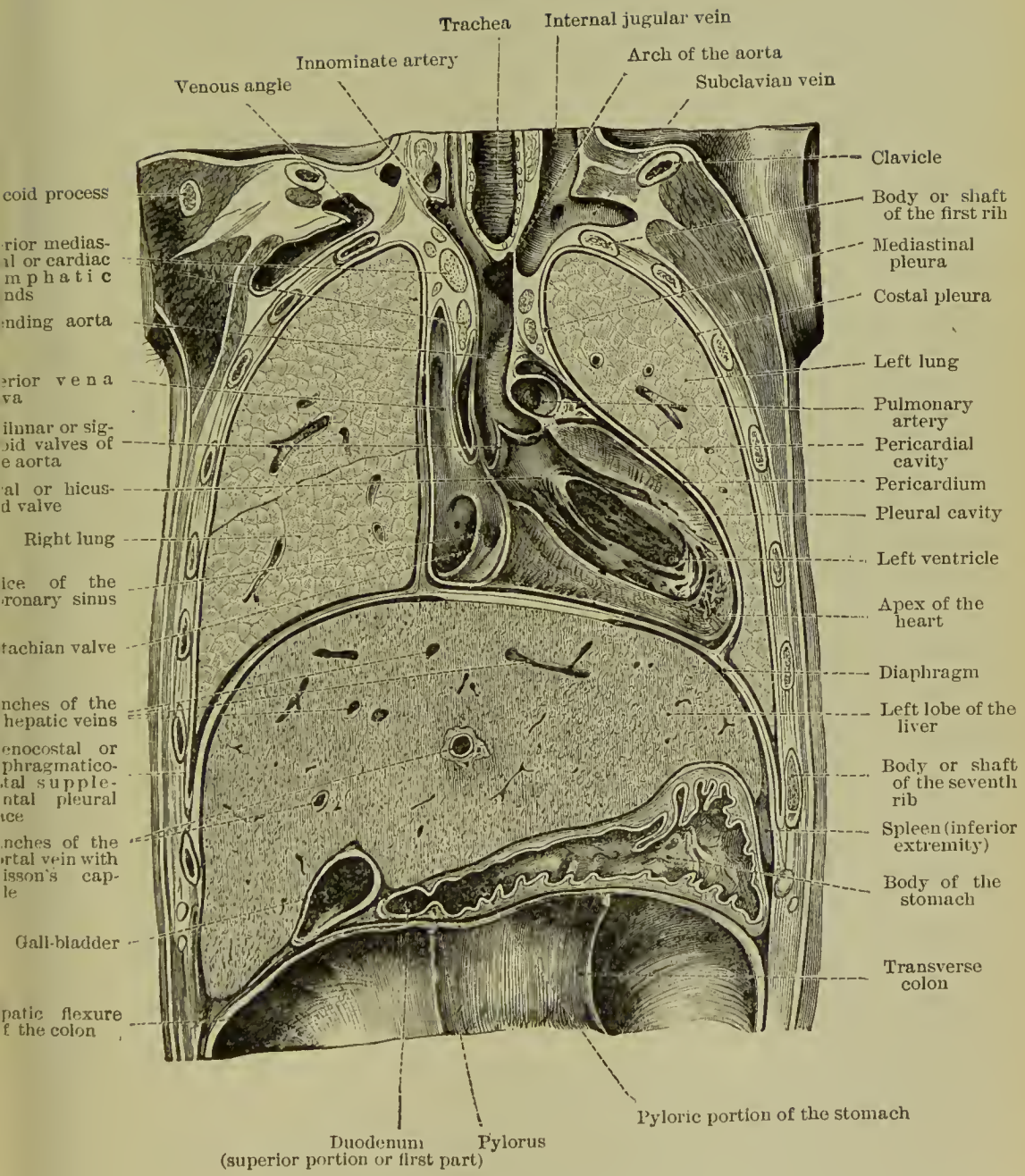

Fig. 19.—Frontal section through the trunk.

abdominal fat is largely decreased also in hyponutrition. This, therefore, favors a loosening of the viscera from their attachments, a downward displacement of the diaphragm, and, thereby, leads to a decrease of pressure in the thorax.

Movability of the abdominal organs and of the heart is also observed in chlorosis. Hoffmann considers this the explanation of the passive dilatation after exertion so frequently reported by some investigators (probably from erroneous observations), but he is probably incorrect. For as we know that the chlorotic has a flaccid muscular system, and that under these circumstances a displaced stomach readily dilates, so the same may be assumed of the heart. In this case we would have a complication of abnormal displacement and dilatation.

We have little definite knowledge of the pathologic processes which are influenced by the amount of space in the thorax and abdominal cavity. Some clinicians (Curschmann, Braun, and Cherchewski) maintain, from their experience, that arterio-sclerosis particularly involving the aorta increases movability, while Determann was unable to confirm this.

It is easily understood that just as a neoplasm burdens the stomach, or hydronephrosis produces a tendency to displacement, so the same conditions arise in a hypertrophied heart. An enlargement of the left ventricle may become so extensive that dilatation of the right ventricle escapes observation (Fränkel). Individual influences play a great rôle, and often a hypertrophied heart shows no displacement.

As a rule, displacement is readily proven by palpation and percussion.

SYMPTOMS

As to the symptoms, there is no unanimity among clinicians. Some, such as Rumpff and Leusser, incline to the view that certain symptoms distinctly indicate cardiac displacement, wandering heart. Persons with marked displacement of the heart cannot sleep for any length of time upon the left side without distress arising. Symptoms become noticeable, such as palpitation, oppression, difficulty in respiration, attacks of syncope, fear, irregularity of the pulse, etc. Other clinicians, such as Braun and Romberg, deny this, and quite properly emphasize that many persons have a greater or less displacement of the heart without any symptoms. Moreover, the persons who usually suffer from wandering heart are exceedingly nervous, and often complications are present, such as alcoholism, cardiac asthenia, etc., to which the symptoms are attributed; this makes an opinion exceedingly difficult.

In the main, I agree with the latter view, and in this connection I wish to reiterate what I have said regarding floating kidney. A healthy person will usually feel no discomfort from the displacement of a kidney or of the heart, although it cannot be denied that displacement of these

organs, when made known to him, and particularly at the onset, may give rise to symptoms. The rarity of cardiac symptoms in healthy women after labor, during which the heart is especially prone to be displaced, favors this view. Moreover, I recently saw a marked displacement of the heart during an obesity eure, while, simultaneously, the symptoms referable to it disappeared. The case was that of a woman, aged 32, having a height of 1.7 meters, and weighing 98 kilograms; the obesity had particularly increased during the last few years. The heart at first revealed normal dulness, the pulse during rest in the sitting posture was from 80 to 88, but upon the slightest exertion it rose to 130, and at the same time decided dyspnea appeared. Questioning elicited the report that, for many years, the patient had slept only upon her back or in the right lateral position. I instituted a moderate hyponutrition, and assured the lady that after the loss of 16½ to 22 pounds she would be able to sleep upon the left side. When, after six weeks, she had lost 20 pounds, quite a decided displacement of the heart to the left could be determined, about 4 to 5 cm., which had not previously been noticeable. Of course, I did not tell her this. General bodily aetivity, as well as that of the heart, had inereased, the pulse during rest did not exeeed 80, and even upon exertion dyspnea did not so rapidly appear. Above all, the lady assured me that she could now sleep the entire night upon her left side without any inconvenience.

Here, therefore, the antifat cure produced such an improvement in the circulation that possible abnormal sensations due to displaeement of the heart were unnoticed by the patient.

In this respect erroneous opinions may easily be formed beeause undernutrition is not well borne by nervous patients. In men who work under mental strain, and in women who have many household cares, it is apt to produce nervous disturbances, while, after they are relieved from their burdens and pressing duties, and are sent away to a pleasant environment, most of them bear a loss in weight of from 11 to 16½ pounds without the consequent displacement of the heart having any unpleasant effect.

Upon the basis of these views, the removal of the symptoms of wandering heart by plentiful nutrition might be supposed the eorrect treatment. But, generally speaking, this is not the case. We must not forget that in some persons blood formation is immoderately inereased by plentiful nutrition, and thus higher blood pressure arises. We do not know under what circumstances cardiac hypertrophy occurs, but it must all the more be reckoned with since the development of this affeetion usually escapes observation. It is quite likely that disturbances regarded as "cardiac neuroses" mark its onset, usually designated eardiae hypertrophy following "luxury consumption." In practice, even after minute investigation of the heart, we may sometimes doubt whether certain symptoms are due to an increased displaeement or to a beginning hypertrophy. Only in

thin, debilitated individuals does hypernutrition appear to be justified. The efforts of the physician must be chiefly directed to combating the nervous symptoms by means of rest, a proper regulation of the muscular activity, plentiful amounts of fresh air, and, perhaps, also the employment of hydrotherapeutic measures. In some cases the binder advised by Abée in Nauheim, a so-called heart brace or heart bandage, may be advised. The chief value of the apparatus is, however, merely suggestive; at all events, unlike the advantage from the employment of the binder in displacement of the abdominal organs, it is impossible to explain by purely mechanical laws the benefits from its use.

DISEASES OF THE PANCREAS

SYMPTOMATOLOGY OF THE DISEASES OF THE PANCREAS

By L. OSER, Vienna

HISTORY

Although physiology and experimental pathology were busy in the last third of the preceding century with a study of the pancreas which has led to a better understanding of the various vital functions of this organ, practical medicine has utilized this knowledge to but very slight extent.

Hospitals and medical colleges, especially the departments of surgery, have devoted increasing attention to the pancreas within the last few years, and it has come to pass that, in making a diagnosis of digestive disturbances from certain alterations of metabolism, the pancreas also has been considered, yet in the popular current of professional life, diseases of the pancreas have been almost ignored.

Regrettable as this apparent apathy is, its cause is easily understood when sought for. Chief among the reasons is the fact that, until recently, this indifference was manifest also in the schools, in the clinics, and in the laboratories. Even to-day, adequate attention is not devoted to the pancreas, as is evident when we consider the manifold and complicated questions brought up for consideration, questions which often relate to elementary but most significant processes. Even pathologic anatomy has added little to our knowledge of the pancreas; at autopsies, the pancreas frequently is not considered at all, or but superficially investigated, and a cursory examination of this organ is usually without value. It is true the difficulties are great. An apparently normal pancreas will, upon careful microscopic investigation, frequently show changes which are exceedingly difficult to interpret correctly for the reason that the organ, during the death agony and, perhaps, also for some time after death, undergoes change from continued self-digestion, and this may very readily be mistaken for a pathologic alteration which had occurred during life.

In the last few years a decided change for the better has taken place, as is shown by the increasingly rich literature. Since the important researches of v. Mering and Minkowski, which point with certainty to the connection between diabetes and the pancreas, an active interest in this

organ has been awakened and, owing to the vietorious advanee of surgery, following the initiative of Gussenbauer, the pancreas has become the border-land between internal medicine and surgery, and incontrovertible proof has been furnished of the great practical importance of this organ.

The pancreas to-day, on account of individual causative observations, is not only the subject of diseussion in inaugural dissertations, but prominent exponents of theoretical and practical medicine in different countries have devoted their best endeavors to the development of a positive foundation for the study of diseases of the pancreas. Naturally we are still far from the goal—but a few important facts have been ascertained, which are not only interesting to the practical physician, but the knowledge of these he cannot and dare not ignore in his professional work. This is our justification for calling elose attention to the present status of clinical knowledge concerning diseases of the pancreas.

This article will be devoted to a description of the *symptoms* by which we are enabled to make a diagnosis of disease of the panereas.

ANATOMY AND PHYSIOLOGY

Before entering upon my actual theme, I desire to emphasize a few points in the anatomy and physiology of the pancreas which have a direct bearing on our subject. First in importance, the gland, as a rule, has *two ducts,* a fact which is not always borne in mind, even by physiologists, since in the animal usually experimented upon—the dog—two ducts are invariably present. The tying of *one* duct, or the introduction of a cannula into *one* duct, may lead to error. In the dog—as recently reported by Helly—these ducts are always distinctly separated, which in man is the exception. Helly, under the direction of Zuckerkandl, has recently studied this subject thoroughly. He examined 50 cases, and among these he only once found the ductus Santorini and no duetus Wirsungianus; in all of the other eases he found the ductus Santorini connected with the ductus Wirsungianus, or the duct existed alone, the latter condition being much the rarer. Of the 50 eases, Santorini's duet had free passage into the intestine in 40 cases, and was obliterated in 10.

The relations between the pancreas and the ductus choledochus are of the utmost practical importance; the latter enters a groove upon the side of the gland, bends toward the duodenum, and, as Zuckerkandl reports, embeds itself, but soon terminates in a canal. Helly, in his investigation of 70 cases, confirmed these reports of Zuckerkandl. He found that the terminal portion of the ductus choledochus is always closely connected with the head of the pancreas to the extent of from 2 to 7 cm.

Pawlow made some interesting experiments in regard to innervation. According to this author, the vagus is the secretory nerve of the pancreas, but this nerve also carries fibers which have an inhibitive secretory func-

tion; in the sympathetic Pawlow also determines secretory and vasoconstrictory fibers. More recent investigations by Wertheimer and Lepage have demonstrated that, after the severing of all nerve centers, secretion does not cease; that, therefore, there must also be an automatic nerve apparatus present in the gland. These investigations permit us to conclude with certainty that in pathologic cases disturbances in secretion may also be caused by disturbances of innervation.

The incessant labors of physiologists in the last few decades have frequently demonstrated the great importance of the physiological functions of the pancreas. Undoubtedly the pancreas is one of the most important organs of the body; in digestion and in metabolism perhaps the most important. The pancreatic juice is the only glandular secretion which is capable of converting all substances that can be at all digested into that form which is necessary for their complete utilization as food. It is more active than any other ferment (trypsin) in the splitting of the albumin bodies; it emulsifies and splits the fats so that they are capable of absorption (steapsin); it transforms the carbohydrates into sugar (pancreatic diastase) and also possesses a milk-coagulating ferment. The secretions of the other digestive glands also possess these properties, but they are not combined in the secretion of any *single* organ. The specific property of the pancreas, which belongs to it alone, is the *splitting up of fat.* In the intestine, it is true, a certain degree of fat-splitting is possible, but only by bacteria. The enormous importance of the pancreatic juice in digestion has been decisively proven by animal experiments, to which we shall later revert. But its *digestive* function is, however, not the only activity of the gland. It also performs an important part in the human economy by the transformation and preparation of sugar. We attribute this function to the *internal secretion,* the conclusive, experimental proofs of which we shall later discuss minutely.

This sketch indicates the great importance of the pancreas in the human economy. Disease of this organ must, naturally, produce severe disturbances, and we might suppose that, in consequence, such marked symptoms would appear that the recognition of a disturbance in function would present no difficulty, but the contrary is the case. How can this fact be explained? The most significant reason is this—that only very extensive or complete destruction of the organ and the occlusion of both ducts will cause characteristic symptoms. In partial disease, or the occlusion of *one* duct, as we shall see, the internal as well as the digestive function may remain intact, and the normal remainder of the gland, although with only *one* duct, and perhaps vicariously, may compensate for the functions of the neighboring organs. Total or very extensive disease is certainly much rarer than partial, and the latter usually gives rise only to doubtful symptoms which are but uncertain points of support for

its recognition. The symptoms are interesting, because, as a rule, it is not only the pancreas that is diseased, but a simultaneous disease of the neighboring organs may be the cause, or consequence, of disease of the pancreas, and its physical signs be much more distinctly and obviously apparent than those of the diseased pancreas. If, for example, chronic pancreatitis develops in the course of gall-stone disease, cholelithiasis forms the most prominent symptom, and the signs of disease of the pancreas may be completely disguised.

The **symptoms of absence of function**, therefore, are of paramount importance in diagnosis, and to these we shall devote our attention.

SYMPTOMS

First among these are *diabetes* and *alimentary glycosuria.*

Autopsy findings long ago directed attention to the relation between the pancreas and diabetes. The oldest, most reliable report was in 1788 by Cowley, who, in a diabetic, found numerous calculi in the substance of the pancreas. Chopart later made a similar observation. Bright, in 1833, saw, in a diabetic, aged 19, a hard, nodular tumor at the head of the pancreas while the gland itself was atrophic. Since that time, similar cases have been frequently mentioned in literature.

The frequent coincidence of diabetes and disease of the pancreas caused Frerichs, Seegen, and Friedreich to assume a causal connection between the clinical and anatomical findings; but it was still questionable whether disease of the pancreas was the cause or the consequence of diabetes. French authors—above all, Bouchardat—took a more decided stand; they proposed a clinical type, diabète maigre, and looked for its foundation in a disease of the pancreas. This assumption was enticing, and was also frequently confirmed by conclusive reports of cases; nevertheless, it remained but a hypothesis until by animal experiment v. Mering and Minkowski finally furnished the incontestable proof that by removing or destroying the pancreas in a dog severe diabetes occurred. A radical change in opinion was the result, and it is now believed beyond all doubt that diabetes in man is caused by disease of the pancreas. The results obtained by these authors were soon confirmed by many others, and in an entire series of animals diabetes was produced by extirpating the pancreas. But diabetes occurred only by removal of the *entire* gland, or, at least, of its *greater portion*; *partial extirpation* gave varying results. If one-fifth to one-fourth of the gland were allowed to remain, v. Mering and Minkowski frequently found no glycosuria. But severe diabetes might also be produced. This depended particularly upon the composition of the portion of the gland remaining. If this was subsequently destroyed by consecutive inflammation or atrophy, diabetes developed; cases of diabetes of medium severity, or only a transitory or more or less permanent alimentary

glycosuria arose; or, as already mentioned, in the great majority of cases no form of sugar excretion took place. Sandmeyer found, in a patient in whom severe diabetes developed 13½ months after partial extirpation of the pancreas, that diabetes persisted for eight months and until death occurred. In the experiments in partial extirpation conducted by Katz and myself, it was impossible to produce severe diabetes, but only *transitory* and *alimentary glycosuria*; in some cases, sugar was absent from the urine.

Minkowski definitely proved that the cause of diabetes in extirpation of the pancreas could be found neither in a lesion of the nerves nor in injuries, and that it could not be attributed to the absence of the pancreatic secretion.

This was demonstrated in an indisputable manner by the transplantation of portions of the pancreas under the abdominal skin, first by Minkowski and later by Hédon. A portion of the pancreas may be detached and, without injuring the vessels which supply it, may be transplanted under the skin of the abdomen. If the portion remaining in the abdominal cavity is removed, no glycosuria occurs, not even the alimentary form; if the section underneath the abdominal skin is subsequently removed, severe diabetes is produced. If the transplanted portion is badly nourished, mild diabetes may occur. If complete atrophy takes place in the grafted portion, severe diabetes develops. The mere tying of the ducts never leads to diabetes, except when such a change due to these processes subsequently occurs in the glandular substance that it is completely destroyed.

It is absolutely true that removal of the pancreas in quite a number of animals has produced diabetes. Besides its digestive function, the pancreas unquestionably has another which is related to sugar metabolism. We do not err when we refer this function to a secretion quite distinct from the digestive juice; this is called the *internal secretion,* or, following Hansemann, the positive function. In what manner this internal secretion exerts its activity is still doubtful. Minkowski, and also most authors, assume that the pancreas in the preparation of sugar also generates a product which acts in the organs.

A series of hypotheses have been proposed in explanation, but no definite proof of the correctness of one or the other is yet at hand.

The hypothesis of Lépine is most interesting; he assumes a glycolitic ferment which is normally produced in the pancreas. This glycolitic ferment reaches the lymph, thence the blood, particularly the white blood-corpuscles, and carries on the preparation of sugar in the tissues. If this ferment is absent, hyperglycæmia and diabetes result. Lépine later modified this hypothesis, no longer referring the point of attack of the glycolitic ferment to the blood but to the tissues. In consequence, as Minkowski points out, the assumption of such a ferment is no longer impossible, although we have no actual proof.

Other hypotheses, such as those of Chauveau and Kaufmann, in which an increase of sugar formation in the liver is assumed to be the basis, and also the views of the brothers Cavazzani are disproven by the experiments of Minkowski, who demonstrated that in experimental panereatic diabetes not an inerease of the sugar production, but a decrease in its consumption, oecurs. We only know, therefore, that the pancreas has a funetion which regulates the eonsumption of sugar, but we do not know the nature of this funetion.

An attempt has lately been made to discover the region in which this internal secretion is found. Langerhans in 1869 described structures in the panereas whieh differ from the pancreatie tubules. These so-called intertubular cell elumps are also found in man and in various animals, and have different forms. Reeently the property of furnishing the internal seeretion has been aseribed to these intertubular eell elumps. Laguesse as well as Diamare some years ago expressed the view that these cell elumps bear some relation to the internal secretion.

Recently Walter Sehulze has attempted to solve this question experimentally. He ligated small portions of the panereas in guinea pigs, and studied the changes whieh followed. After a few days an atrophic process developed in the glandular elements which continually inereased—even after 80 days, at whieh time the tubular area was eompletely atrophied, being replaced by delieate eonneetive tissue; the eell clumps had taken no part in this atrophy, but, on the eontrary, showed not the slightest ehange. Schulze coneludes from this that these cell clumps are substantive struetures independent of the glandular system of the pancreas. It is true this does not prove that they have any bearing on sugar metabolism. It had long been believed that these struetures were in their nature different from the seereting glandular elements. Langerhans himself regarded them as nervous elements; other authors have looked upon them as lymphatie tissue. Some authorities believe them to be embryonal remains, and they have also been considered in conneetion with the seeretion of the diastatic ferment.

Walter Schulze's experiments are evidently of importance, but the proof that these struetures do not belong to the glandular system and are related to the internal seeretion must be furnished by further investigation. Szobolew recently undertook to prove by chemical and microscopical investigations that Langerhans's cell elumps have some relation to sugar metabolism, and decided this to be a fact. In two cases of panereatic diabetes the disappearance of these cell clumps was confirmed. Opie arrived at the same conclusion; in 11 cases of interlobular panereatitis in whieh form of the disease Langerhans's cell elumps are visible only when the process is very far advanced, he noted mild diabetes only once, and at that in a case in which the sclerosis had also attacked Langerhans's cell clumps. In three eases of interacinous panereatitis in whieh form also the previ-

ously mentioned cell clumps were implicated, diabetes was present twice, and in the fourth case of diabetes there was complete hyaline degeneration of Langerhans's islands.

If we succeed in proving positively that these cell clumps are concerned in the internal secretion, important knowledge as to the true cause of pancreatic diabetes will have been gained.

The demonstration in animals of an experimental pancreatic diabetes enables us to understand much more fully than was previously possible the numerous autopsy reports in literature of changes in the pancreas in the diabetes of man. All doubts disappear, and it is certain that in a large number of cases disease of the pancreas was the cause of the diabetes. At the necropsy of diabetics various changes were found in the pancreas, most frequently atrophy, but often also induration, calculi formation, carcinoma and fatty degeneration. Doubtless, some of these changes in the pancreas are not the cause of diabetes; in many cases they are either its consequences or are in no way related to it. Atrophy has often been demonstrated to be the cause of the marasmus which occurs in diabetes. Hansemann reports a characteristic form of *diabetic atrophy* with well developed symptoms of an active process added to the process of the secretory cells; this belongs to the varieties of interstitial inflammation, similar to that in certain forms of granular atrophy of the kidneys. Hansemann describes early stages of these processes, and designates them as anatomical individualities which must necessarily lead to diabetes.

If, in many cases of diabetes, no changes are found in the pancreas, not even upon minute microscopic examination, this is additional proof that there are other causes of diabetes than disease of the pancreas—a view now generally accepted. Lanceraux in the last Congress at Paris maintained the unity of diabetes, and of its origin in disease of the pancreas. He assumes that there are anatomical determinable changes or functional disturbances of the pancreas which lead to diabetes. Lanceraux formulates a hypothesis against which weighty objections may be raised, and which cannot be sustained at this present time.

A vulnerable point in the theory of pancreatic diabetes in man is the fact that *total destruction* of the pancreas has been found without diabetes having existed during life. Hansemann's hypothesis does not sufficiently explain the fact that in cases of total destruction of the pancreas by diffuse carcinoma, carcinomatously degenerated cells may perhaps furnish the internal secretion of the pancreas, as there are also other varieties of total destruction of the pancreas without diabetes. Perhaps future investigation of the anatomical findings revealed in Walter Schulze's experiments will clear up the situation. No matter what the conclusion may be, this loop-hole cannot alter the fact that in man diabetes is due to the absence of the pancreatic function.

Not only permanent diabetes but also *transitory and alimentary glyco-*

suria may be caused by changes in the pancreas; this is shown by animal experiments, as well as by the facts demonstrated in the disease in man. After various injuries of the pancreas, even although slight, transitory glycosuria may occur. In 15 out of 32 cases of partial extirpation, Minkowski saw transitory glycosuria. Minkowski, as well as myself, demonstrated alimentary glycosuria in the previously mentioned experiments. In one of these there was a transitory excretion of sugar after every operation upon the pancreas. Upon an exclusive meat diet, on the administration of 10 grams of grape sugar slight glycosuria occurred, and after 50 grams it was marked. In an interesting article Wille has furnished proof that alimentary glycosuria in man is also connected with changes in the pancreas. In 800 patients in the Hamburg General Hospital, Wille made feeding experiments, administering 100 grams of grape sugar in each case, and, with a positive result, this was repeated once weekly. In 47 cases he found alimentary glycosuria. In 77 of the 800 cases an autopsy was held; in 15 of these alimentary glycosuria had been recognized during life, and at the autopsy high-graded changes in the pancreas were invariably found. Ten doubtful cases still remained in which during life alimentary glycosuria had been proven, and at the autopsy marked changes in the pancreas were found. Alteration in the pancreas was also observed at the autopsy in cases which, during life, had given no evidence of alimentary glycosuria. Wille arrived at the conclusion that the regular appearance of alimentary glycosuria is an important diagnostic sign of existing disease of the pancreas, that in a periodically appearing alimentary glycosuria the suspicion of disease of the pancreas should not be at once rejected, but that, in the absence of this symptom, the pancreas cannot be looked upon with certainty as normal.

These reports of Wille are noteworthy; they show that cases of *constant alimentary glycosuria* may be the result of changes in the pancreas. But the relatively small number of positive reports prevents our proving with certainty that this is always the case. Alimentary glycosuria—also the constant form—is frequently found associated with various diseases, particularly those of the nervous system. In these numerous cases but few autopsy reports relating minutely to the pancreas are at hand, and it may be assumed that there are cases of alimentary glycosuria not caused by changes in the pancreas. It may be true, as in permanent diabetes, that constant alimentary glycosuria occurs without changes in the pancreas. In spite of these objections, we may maintain, on the basis of our present knowledge, that the proof of permanent diabetes as well as of constant alimentary glycosuria justifies us in including changes of the pancreas in our diagnostic calculations.

A second group of symptoms develops from the absence or by the destruction of the *digestive function*. We know that the pancreas has a

fat-emulsifying and fat-splitting, a proteolytic and an amylolytic, function. Disease of the pancreas or its destruction must necessarily lead to disturbances of this kind. The fact is also operative that a large portion of the gland, or almost all of it, must be incapable of performing its function, hence symptoms due to the absence of function must appear. Add to this the circumstance that, as a rule, two ducts are affected, in some animals even more, and it becomes clear that if only *one* duct be occluded, provided that in the other the secretion is sufficiently active, no digestive disturbances may follow. This explains why we so rarely observe disturbances of digestion as a result of disease of the pancreas.

Let us first consider the absence, or the disturbance, of *fat digestion*. This is shown either macroscopically, or only microscopically or chemically in the changed condition of the feces.

For a long time isolated clinical observations have pointed out the connection between impaired assimilation of fat and changes in the pancreas, and an attempt was made to prove this experimentally in animals. Even Claude Bernard was of the opinion that pancreatic juice splits the neutral fats, emulsifies them, and is therefore necessary for the absorption of fats. Among those who differed with him was Schiff. Opinions varied until the discovery of experimental panereatie diabetes by v. Mering and Minkowski, and the digestive disturbances following the removal of the pancreas were studied. Although even to-day there are diversities of opinion and many doubts concerning these points, nevertheless the view that, in the absence of the pancreatie function, disturbances arise in the absorption of fat is generally accepted. Abelmann, a pupil of Minkowski, found that with an absence of the pancreas non-emulsified fat is not absorbed at all, and emulsified fat only to a slight extent; only in the case of cream was the absorption more complete. With partial extirpation, 50 per cent. of emulsified fats was absorbed and 80 per cent. of milk. Upon the addition of pancreas to the food, digestion improved. Fat-splitting, even with a total absence of the pancreas, was undisturbed. Similar reports were made by Sandmeyer, Cavazzani, and Baldi. Very different results were obtained by Hédon and Ville who raised objections to Abelmann's method of research, and in this they were supported by Pflüger. Hédon and Ville found that after total extirpation of the pancreas, fat absorption continued, although to a lessened extent; in a case of severe diabetes it amounted to about 18 per cent.; fat-splitting remained about normal, principally free fatty acids, but also small amounts of soap. In more extensive investigations Siegfried Rosenberg ascertained that in experimental, gradual destruction of the parenchyma of the gland, fat absorption still showed almost normal values; that, however, upon increasing glandular destruction, it is damaged. In regard to fat-splitting, which Rosenberg attributes mainly to bacterial action, he believes that the low fat-splitting figures found with increasing glandular destruction are due

to the frequent evacuations, and are caused by the undigested material which passes through the intestine.

In complete unanimity with the results of animal experiments, clinical experience teaches us that frequently, but by no means always, in disease of the pancreas, in chronic inflammation, in the case of neoplasms, of cysts, of stones in the ducts, and in atrophy, a deficient digestion of fat can be proven. The first undoubted report is by Kunzmann in 1820; he saw a profuse discharge of fat in the feces of a man who suffered from induration of the pancreas with obliteration of Wirsung's duct, chronic jaundice and dropsy. A report by Fles is very interesting. A diabetic who had eaten considerable bacon and fat meat discharged ounces of fat in his feces that could be separated from the stool. If the patient took with his food an emulsion prepared from the pancreas of a calf, the fat disappeared. At the autopsy high-graded atrophy of the pancreas was found. In a case of carcinoma of the head of the pancreas observed by me, there were present for months uncommonly copious, thick, pappy, fecal discharges which were always profusely admixed with fat. In both cases jaundice was absent—a fact which must be particularly emphasized, for, according to the excellent researches of Friedrich Müller, if the bile is absent from the intestine fatty stools are quite normal.

The forms of disturbance of fat digestion which are combined with disease of the pancreas vary considerably. They are sometimes recognized macroscopically, and are then designated as *true steatorrhea.* In this condition large quantities of oily or fluid, yellow and yellowish-brown, fatty masses are discharged with the evacuations, or even independent of them. In the cold these become compact, resembling butter, grease or wax; the hardened fat may then completely envelop the feces contained within it. Pfibram, in a case observed by him, closely analyzed these fatty discharges, and proved their similarity to the varieties of fat ingested. Chemical examination of the fatty masses which are visible to the naked eye shows neutral fats and fatty acids.

In diseases of the pancreas the stools are of the consistency of clay, grayish-white, or colorless, occasionally asbestos-like, and the increased amount of fat can only be recognized by chemical and microscopical processes. Unquestionably there are many transitional stages between true steatorrhea and the previously mentioned *fatty stools,* so that the difference between the two forms of dejecta may be assumed to be a graduated one. The chemical investigation of these feces chiefly reveals neutral fats, fatty acids and soaps. Such dejecta, rich in fat, are found by no means exclusively in diseases of the pancreas, but may also occur normally in persons who have eaten such a great amount of fat that the intestine is incapable of absorbing it; in the majority of cases, however, the condition is observed when bile is absent from the intestine, in certain diseases of the intestine, in amyloidosis, in tuberculosis of the intestine, in distributed atrophy of

the mucous membrane of the small intestine, in cascation of the mesenteric glands, in chronic tubercular peritonitis, and perhaps—to which also Nothnagel calls attention—in intense catarrhal processes. It is evident from this fact that a high percentage of fat in the stools does not prove disease of the pancreas, and we must first exclude all other causes of insufficient fat absorption before we can regard an affection of the pancreas as the cause of the disturbed fat digestion.

Müller, who was positively unable to determine a greater amount of fat in the stool upon the absence of pancreatic juice from the intestine, designated *diminished fat-splitting* as a factor of disease of the pancreas. Müller excised the pancreas of a dog, and, under antiseptic precautions, placed it in sterilized milk in the incubator for 24 hours. Fatty acids amounting to from 42 to 45 per cent. were present. In a control experiment in which the pancreatic ferment was destroyed by boiling, no fat-splitting occurred. After Müller had previously proven by experiment that intestinal bacteria had but a slight influence upon fat-splitting, he concluded that the important factor for fat-splitting in the intestine is contained in the pancreatic juice, and the absence of the pancreatic function is expressed by disturbed fat-splitting. In a series of cases of pancreatic disease, Müller proved the disturbed fat-splitting with certainty, his reports being confirmed by several authors, but denied by others.

The question has been most thoroughly studied by Katz. He formulated an expeditious method which enables us to determine the degree of fat-splitting in 24 hours. In the examination of a number of cases observed in my hospital, in which during life reduced fat-splitting, and at the autopsy pathologic changes in the pancreas, were determined, he came to the conclusion that a diminution of fatty acids and soaps to below 70 per cent. of the total amount of fat in the fatty stool favors a diminished or even completely arrested function of the pancreatic juice; but in nurslings, and in profuse diarrhea, this decrease had no significance. Other authors have also noted disturbed fat-splitting in pancreatic disease, for example, v. Noorden, Rosenheim, Anschütz.

Diminished fat-splitting in disturbed pancreatic function may, therefore, be considered as proven, and the validity of this proof does not suffer from the fact that there are undoubted cases—perhaps many—in which no disturbance of fat-splitting can be determined; even in widely distributed disease of the organ, a healthy portion of the gland may serve the excretory ducts, which may discharge a sufficiency of pancreatic juice into the intestine.

Where a disturbance of fat-splitting has been found without disease of the pancreas being determined, the circumstances are different. Katz specified that in nurslings and in profuse diarrhea reduced fat-splitting might be found without coexisting disease of the pancreas. Certainly in all cases in which an increased peristalsis rapidly propels the contents

through the intestine, the pancreatic juice, normal in amount, will not have sufficient time to split the fat to such an extent as is possible under normal circumstances.

Zoja, who recently published a comprehensive study of fat absorption, mentions two cases of reduced fat-splitting without changes being found in the pancreas or in the excretory ducts. In one case there was stenosis of the large intestine, and in the other carcinoma of the gall-bladder. Zoja recognizes in deranged fat digestion an important indication of an affection of the pancreas, but, on account of the great number of reported cases he arrived at the conclusion that the disturbed fat-splitting is not the most important symptom, but the small amount of soaps which he invariably demonstrated in diseases of the pancreas. Deucher had previously called attention to the importance of this factor. The demonstration in the feces of a decided quantity of neutral fats, or of a great amount of fatty acids, particularly, however, of a deficiency in soaps, according to Zoja, points with great likelihood, if not with positive certainty, to the absence of pancreatic juice from the intestine. The main stress is placed by Zoja upon the lessened quantity of soaps. The greater the percentage of soaps, the more certainly may an occlusion of the pancreatic ducts be excluded; the less the quantity of soaps in proportion to the neutral fats and fatty acids, the more readily may occlusion of the pancreatic duct be assumed. The less the amount of pancreatic juice which flows into the intestine, the slighter is the amount of alkali, and, therefore, also the smaller the amount of soaps. In this reasoning Zoja finds no difference between the animal experiment and observation in man, which has also been emphasized from the standpoint of fat-splitting, since Abelmann, as well as Rosenberg, found smaller amounts of soaps.

We cannot enter here upon a criticism of the work of Zoja. I shall only mention that Abelmann reported the frequent finding of quantities of soaps which reached the normal. Müller previously objected to this method of determining soaps and the combined fatty acids, and designates the proportion of free fatty acids to the soaps in the feces as an inconstant one which depends upon accidental conditions. I must also state that in a case of Zoja's of recognized pancreatic affection, normal values for soaps were found. The relative value of the factor emphasized by Zoja, the lessened soap figures in proportion to the large amount of neutral acids and fatty acids, cannot be denied; but more complete investigations are necessary finally to solve the important question of the diagnosis of diseases of the pancreas. We can only state positively that disturbed fat digestion is an important symptom in pancreatic disease. An increased amount of fat in the stools is alone not a basis for the assumption of a pancreatic affection. If no jaundice exists, and no disease of the intestine is present, if increased peristalsis by which the ingesta are rapidly propelled through the intestine does not explain the insufficient fat digestion, then the sus-

pieion of disease of the panereas is eertainly justified. Some forms of altered fat digestion, true steatorrhea, in whieh larger, even maeroscopieally recognizable, amounts of fat are passed with or without the stool, whieh show diminished fat-splitting with a eertain inereased amount of fat in the stools, and low quantities of soaps in proportion to the fatty acids and neutral fats, are faetors whieh point with great likelihood to disease of the panereas. It ean only be positively proven, however, when other symptoms due to absenee of funetion, sueh as diabetes or insuffieient nitrogen absorption, or eertain elinieal symptoms, sueh as tumor, bronzing of the skin or pancreatie eolie, are present.

Equally important is the *absence or disturbance of albumin digestion,* whieh is observed experimentally and elinieally in diseases of the pancreas. Abelmann found that when the panereatie juiee is absent, only a portion of the proteids is absorbed; this averages 44 per eent. in animals in whom the panereas was removed, and 54 per eent. in the eases of partial extirpation of the panereas. If, on meat diet, pig's panereas was simultaneously given, the absorption of nitrogen was deeidedly inereased, and 74 to 78 per cent. was absorbed. After a meat diet the presenee of numerous maeroscopieally recognizable undigested musele fibers was eonspieuous. Similar results were obtained by de Renzi, Cavazzani, Sandmeyer, and Harley. In an instanee of total extirpation of the panereas performed by Katz and myself, and whieh was followed by diabetes, after meat was taken for the first time, large portions of undigested meat were maeroseopieally found in the feees. Under mieroseopie investigation numerous musele fibers with distinet transverse striæ were deteeted.

Rosenberg's investigations were most thorough; after tying the exeretory duets, injeeting into them an aeid solution, and subsequently tying again, as is done for the gradual destruetion of the glandular parenehyma, he found at onee that the absorption of nitrogen was but slightly altered, even before he eould determine any disturbanee in the absorption of fat and earbohydrates. Later, after the removal of the degenerated glands, nitrogen metabolism fell to 33 and 35 per eent., while 41 per eent. of fat and 50 per eent. of earbohydrates were still being absorbed.

Quite as positive is the elinical proof based upon numerous observations that in man disease of the panereas eauses disturbanees of albumin digestion. Deficient meat digestion is most eonspieuous. In the feees there are numerous undigested muscular fibers, as may sometimes be recognized with the naked eye, but mieroseopie examination shows them very distinetly. In the ease of a diabetie, reported by Fles, the dejeeta contained uncommonly numerous, undigested, quite distinct, transverse museular fibers. If the patient took ealf's pancreas with his other food, the meat was again perfeetly digested, and the musele fibers again appeared in the stool when no panereas was added to the food. Harley recognized a similar condition in a ease of pancreatie abseess. v. Aekeren noted impaired

digestion of meat in carcinoma, Küster in a case of cyst, and Lichtheim in a case of calculus formation in the pancreas. In a case of carcinoma of the pancreas that came under my observation, I noted numerous remains of transverse, striped, muscular fibers with distinct muscular structure. Zoja recently reported a case of carcinoma of the head of the pancreas with occlusion of Wirsung's duct and of the common gall-duct, in which numerous fibers of meat were found in the feces with distinctly developed transverse striation. A similar condition was observed by Auerbach in a case of carcinoma of the pancreas, and by Cipriani and Giudiceandrea in pancreatic calculi.

The insufficient nitrogen absorption in pancreatic disease has also been proven by exact researches in metabolism. Hirschfeld conducted a series of thorough analyses in diabetes. He found that 32 per cent. of the nitrogen substance ingested could be found in the feces, while 5 to 6 per cent. corresponds to the norm. It is true this was not proven by autopsy findings. But this proof was present in a case of Weintraud's; 45.2 per cent. of the albumin of the food, and 22.2 per cent. of the fat, were lost. At the autopsy a markedly contracted, indurated pancreas with decided increase of the connective tissue was found, and in this only isolated glandular globules could be seen. Weintraud, in agreement with the results of Rosenberg's animal experiments, lays the principal stress upon the fact that in diseases of the pancreas proteid absorption is more decidedly, or at least as greatly, disturbed as fat absorption; while in intestinal diseases, in which the intestinal apparatus for the absorption of fat functionates insufficiently, as in amyloid disease of the intestine, etc., the absorption of fat is comparatively as much affected as that of the albumin. The conditions were similar in a previously mentioned case reported by Zoja, of carcinoma of the pancreas with occlusion of Wirsung's duct. More than 70 per cent. of the nitrogen of the food was excreted, and hardly 30 per cent. absorbed. Unfortunately, but few reports of experiments are at hand; but even with the data we have, there can be no doubt that this is a positive method for the recognition of diseases of the pancreas.

Recently Sahli has proposed another method for testing the digestive function of the pancreas for proteids. He determined by exact experiments that gelatin capsules hardened with formaldehyd—he calls them glutoid capsules—after reaching a definite degree of hardness, are soluble neither by water nor by hydrochloric acid pepsin digestion, but only so by pancreatic juice (by trypsin). If these capsules are filled with a substance which can be recognized in the saliva or in the urine—best with iodoform—in cases of arrested pancreatic digestion a reaction for iodin does not occur at all, or only very late. With normal motility of the stomach and sufficiently good pancreatic function the iodin reaction in the saliva shows itself in from 4 to 8 hours. According to Sahli, from the absence or from

the delayed appearance of the iodin reaction we may determine a disturbance of the pancreatic function, provided the motility of the stomach is not reduced. These reports of Sahli are very interesting, and if, in a large number of tests, the absence or the late appearance of the "glutoid reaction" coincides with anatomical proof of a pancreatic disease and arrested secretion, we have a positive and infallible sign of disease of the pancreas.

There can be no doubt that the *digestion of carbohydrates* suffers when the pancreatic function is absent or disturbed. Rosenberg has considered this in his animal experiments. So long as the pathologic process in the pancreas is not too far advanced, normal or almost normal absorption was found; when the process had made further progress, the figures decreased decidedly, so that, in this case, digestion was already seriously damaged. In an experiment at the conclusion of which the degenerate glandular remains were removed, carbohydrate absorption as well as fat-splitting sank rapidly and decidedly, with a loss in the absorption of nitrogen and fat; on pancreas feeding, in all the experiments a conspicuous improvement was noted. Clinical reports in regard to this factor are very few. In recent isolated cases, as, for example, by Auerbach and Edoardo Italia, it is expressly stated that considerable starch was found in the stools.

When the pancreatic function is either absent or disturbed, impaired digestion occasionally produces conspicuous symptoms that are often noted by the patient himself. The stools have a *massive appearance* which is out of proportion to the amount of food ingested. I have seen a number of cases in which this symptom led me to suspect a disease of the pancreas, and the further course of the affection justified this assumption. Much of the food passes through the digestive tract without being utilized. *In spite of plentiful food, there is constant loss of weight, and conspicuously massive, thick, pappy or compact dejecta, whose amount appears to be decidedly greater than the ingested food, and this is an important symptom which calls for the closest attention of the physician.* Exact microscopic and chemical analysis explains this massiveness; muscle fiber as well as fat and carbohydrates are excreted without being assimilated.

The symptoms due to absence of function unquestionably furnish the most important aid in the recognition of an affection of the pancreas, and must therefore be first described. There are, however, a few symptoms which cannot be regarded as denoting absence of function, which, nevertheless, enable us to recognize that the pancreas is diseased. Among these we must consider the following: *Bronzing of the skin, peculiar colic and pains of a definite character in the epigastrium; tumor or resistance in this region, and certain forms of jaundice.* These signs, with the exception of tumor formation, only become important when they are combined with permanent or transitory symptoms of absence of function of the gland.

French authors were the first to direct attention to a peculiar form of diabetes with deposition of pigment in the skin—diabète bronzé. This pigment formation is the expression of hemochromatosis, which develops in the skin as well as in the different organs. The color of the skin is brownish, brownish-black, or somewhat grayish, the discoloration is *diffused over the entire body,* and *nowhere are pigmented areas observed, not even upon the mucous membrane,* and this differentiates the condition from Addison's disease.

The cases reported up to the present time are few in number. In the book I published in 1898 I mentioned 22, Anschütz, in 1899, 24 cases. In most instances changes were found in the pancreas. It was sclerotic, rusty-brown, the excretory ducts were patulous. Jeanselme had already declared diabetes to be due to sclerosis of the pancreas which develops as a result of deposition of pigment. Anschütz recently studied an undoubted case. During life, besides the symptoms of diabetes, there were also signs of disturbed secretory function: distinct fat-splitting and fatty stools. For this reason a diagnosis of disease of the pancreas was made. At the autopsy, a chronic, indurated pancreatitis with general hemochromatosis was found. The deposition of pigment caused chronic inflammation of the pancreas, and this in turn the diabetes. A similar report and explanation were furnished by Opie.

Bronzing of the skin diffusely distributed, from which hemochromatosis may be concluded, absence of pigment areas in the skin and in the mucous membranes, and simultaneously diabetes, warrant the assumption of disease of the pancreas, particularly if, at the same time, secretory disturbances, fatty stools, deficient digestion of fat and albumin can be determined.

From the *pain* alone—no matter what form it may assume—disease of the pancreas can never be diagnosticated. In pancreatic affections pain may be of many varieties and degrees, it may be *persistent* or *paroxysmal.* The *former,* sometimes remittent or paroxysmal, or gradually becoming more severe, is found in abscess, in hemorrhage, in acute and chronic pancreatitis, and in tumor. In carcinoma the pain may, under some circumstances, be of extraordinary intensity, so that the patient anxiously avoids any movement, shrinks at a loud word, and rejects food. In combination with a feeling of extreme weakness and marked prostration, this pain shows certain peculiarities such as are very rare in other tumors of the abdomen. Whether pressure upon the celiac ganglion or torsion of the same causes this peculiar pain has not been proven, but it is unquestionably a characteristic feature, and we are justified in entertaining the suspicion that the seat of the affection is the pancreas.

The *paroxysmal pains* may resemble sharp colic limited chiefly to the epigastrium, and, under certain circumstances, may also point to disease of the pancreas. By some authors special stress is laid upon this pain.

Epigastric colic, as such, naturally proves nothing, not even that, for example, as in the case described by Münnich and Holzmann, its main point of concentration is under the left arch of the ribs. We know that renal colic or gall-stone colic, colic of the appendix, and a beginning pericolitis may cause similar pains in the region of the splenic flexure. We are, then, only justified in diagnosticating a disease of the pancreas if pancreatic concrements are found in the feces, or if, after prolongation of the disease, symptoms due to absence of function appear, such as diabetes, or insufficient digestion of proteid or fat. We may conclude from this that the same process that gives rise to prolonged colic also brings about changes in the parenchyma of the pancreas, and gradually disturbs or arrests the function of the organ. In calculi formation there are chronic inflammatory changes with connective tissue proliferation, and these gradually displace and replace the gland substance.

Not only in stone formation does colic occur, but also in all cases of occlusion of the excretory ducts and of the secretory passages in the interior of the gland, as from cicatrices, neoplasms, indurated inflammations or hemorrhages. In cysts of the pancreas these colics play a particularly important rôle. In 104 cases operated upon Takayasu found colic a symptom in 64 cases. As this form of pain is evidently very much more rare in other cysts of the abdomen, its presence, as Leube emphasized, is an important sign of pancreatic cysts. It may be asserted that these colics are much more frequent than is to-day assumed. How many so-called nervous gastralgias may depend upon pancreatic colic! Minute investigations in metabolism and absorption may, perhaps, show transitory or permanent anomalies which occur in the course of a pancreatic affection.

A *feeling of resistance or a tumor* in the region of the pancreas forms a significant symptom. Naturally we must be certain that this resistance, or the tumor, actually springs from the pancreas. This proof, on account of the concealed position of the organ, is difficult to establish. Primarily we must consider carcinoma, cysts, and indurative inflammation, as the acute processes, acute hemorrhagic pancreatitis, abscess, hemorrhage and necrosis are evidently much rarer occurrences.

In a small number of cases of *carcinoma,* in about $\frac{1}{6}$ to $\frac{1}{4}$, a tumor can be felt, but even then the differentiation from carcinoma of the duodenum, from carcinoma of the choledochus and of the porta hepatis, from lymph-gland tumor, from aneurysms of the hepatic artery, from products of inflammation of a tuberculous nature, even from carcinoma of the colon or pylorus, is very difficult, occasionally impossible. The differentiation from tumors of the colon or pylorus is most readily made since pancreatic carcinomata are, as a rule, fixed, while carcinomata of the colon and pylorus are frequently movable. By artificial inflation of the stomach and colon, by the demonstration of symptoms of stenosis in the stomach or

intestine, by the chemical and microscopic investigation of the gastric and intestinal contents, as well as by other clinical signs referable to disease of the stomach, the intestine, or of the pancreas, the diagnosis may occasionally be made with certainty. Much more difficult, almost impossible, is the differentiation of tumors of the duodenum or the biliary passages. Here the appearance of symptoms which indicate absence of function of the pancreas may clear the situation.

The appearance of a *cystic tumor* in the region of the pancreas does not at all prove that we are dealing with a pancreatic cyst.

Echinococcus of the liver, of the spleen, of the mesentery and of the peritoneum, hydronephrosis, dropsy of the gall-bladder, cysts of the omentum or the mesentery, the spleen, the kidney or the adrenals, soft sarcomata of the liver, accumulations of fluid in the bursa omentalis, etc., may give rise to confusion. It would lead us too far afield to enter upon the details of the diagnostic factors; frequently the diagnosis can only be made with certainty at the operation.

The *inflammatory tumor* is rarely recognized during life; usually it is confounded with neoplasms. Often, however, at the operation it is not quite clear whether an indurative inflammation or a neoplasm is present, and only from the gradual disappearance of the tumor, as, for instance, after the operative removal of gall-stones, do we recognize that a chronic inflammation of the pancreas was present, and this appears to be a by no means rare occurrence.

One of the most frequent symptoms of disease of the pancreas is *jaundice*. The intimate relations existing between the head of the pancreas and the common gall-duct sufficiently explain this. Unfortunately there is no characteristic form of jaundice which indicates disease of the pancreas. The form in which the jaundice slowly but steadily increases and finally leads to complete occlusion by compression of the common gall-duct, is also found in tumors of the duodenum, of the hilus hepatis, and in lymph-gland tumors, all of these causing a similar development of the jaundice as well as the Bard-Pic syndrome: Intense, chronic, gradually increasing jaundice with enormous dilatation of the gall-bladder, rapid emaciation and cachexia with usually subnormal temperature, and the absence of decided enlargement of the liver are not confirmatory, even although it cannot be denied that this symptom-complex occurs more frequently in carcinoma of the head of the pancreas than in tumor of the neighboring organs, or in stone formation in the biliary passages. The relatively rare appearance of the cardinal symptoms, of diabetes, of insufficient fat and albumin digestion, may, it is true, here also indicate the correct diagnosis.

Much less convincing are other symptoms which are observed in disease of the pancreas: *Emaciation, digestive disturbances, salivation, vomiting, constipation, hemorrhagic stools, intestinal occlusion, fever.* It is true

a proof of a sequestrating pancreas, or of pancreatic conerements in the stools, may undoubtedly lead to the diagnosis of disease of the organ.

The acute inflammations, hemorrhagic pancreatitis, pancreatic apoplexy and necrosis continually appear with severe symptoms: Sudden, severe and paroxysmal pain in the gastric and umbilical region, nausea, vomiting, immediate and severe collapse, great anxiety, enormous increase in the pulse rate, and not infrequently the picture of acute intestinal occlusion. The differential diagnosis between internal incarceration and the previously mentioned processes is exceedingly difficult, and usually can only be made at the operation or at the autopsy.

In disease of the pancreas some authors lay special stress upon certain changes in the urine which enable us to make a diagnosis of involvement of the pancreas, such as an *insufficient excretion of indican* and the proof of *pentosuria* or *maltosuria*; unfortunately, these views have not been confirmed. During pancreatic digestion, as is well known, indol, the mother substance of indican, develops. For this reason we formerly, with a suspicion of disease of the pancreas, first directed particular attention to the urine. In a case of occlusion of the small intestine Gerhardi concluded from the fact that indican was not increased that disease of the pancreas was the cause of the occlusion; the autopsy confirmed Gerhardi's view. The animal experiments of Pisenti appear to confirm this opinion. In our animal experiments, at my suggestion, my assistant, Dr. Katz, invariably examined the urine for indican and always found this substance increased. Reports are at hand showing that, in the course of disease of the pancreas in man, the amount of indican was also increased.

Just as little has the appearance of *maltose* or *pentose* in the urine proven itself characteristic of pancreatic disease.

In reviewing the important symptoms which have been mentioned in this article, it is apparent that the most reliable aids in the diagnosis are the *symptoms due to absence of function of the gland, and the proof of resistance or of a tumor having its seat in the pancreas.* In a detailed description of the individual forms of the disease, we are convinced by the careful consideration and investigation of the different symptoms in a number of cases that the diagnosis may be made with certainty, or with more or less likelihood. In many—probably in the great majority—of cases, if we go beyond an assumption we find ourselves upon the wrong track. If any result be obtained it is only possible by persistent, tireless investigation. Such a result is not only of theoretic, but also of great practical value, as has been shown by the efforts of surgeons, who, interfering at the right time, have cured many affections which had endangered life, such as chronic and acute inflammatory processes, and neoplasms of the pancreas.

We are now at the dawn of rational treatment of disease of the pancreas, and undoubtedly internal therapy will also produce practical results

when we are able to diagnosticate the by no means rare and curable affections of the pancreas. On account of the intimate relation between the biliary passages, the liver, and intestines on the one hand, and the pancreas on the other, the consecutive changes in the pancreas will unquestionably—I might say will unwittingly—be cured by internal treatment. When we gain a deeper insight into the pathology of the pancreas, and are able to recognize clearly the pathologic changes in this organ, it will perhaps often be possible not only to prevent the implication of the pancreas, but to restore to the normal the already diseased organ in so far as this is at all possible.

DISEASES OF THE LIVER AND BILIARY PASSAGES

JAUNDICE AND HEPATIC INSUFFICIENCY

By O. MINKOWSKI, Cologne

JAUNDICE

GENERAL CONSIDERATIONS

When a patient with jaundice presents himself to our notice, certain peculiarities are conspicuous at the first glance. In a yellow, artificial light the *yellow discoloration of the skin* may easily escape observation, but in broad daylight this is strikingly obvious, and we then perceive the yellow discoloration of the *conjunctival scleræ*; the transudation of the tissues of the lips and the mucous membrane of the mouth with the same yellow coloring matter also becomes visible if we render the tissues bloodless by pressing upon the mucous membrane. This yellow color is readily recognizable upon the hard palate where the greater tension of the mucous membrane makes it pallid.

The yellow discoloration is, in fact, due to an *overflooding of the organism with biliary coloring matter,* as is proven by the composition of the urine which, with its *brownish tint like the color of beer* and the *yellowness of its foam,* shows the presence of bilirubin. The intense staining power of this coloring matter is immediately perceptible on dipping a piece of filter paper into the urine, and is also manifested by the yellow stains which may appear upon the linen of the patient. Certain proof of the presence of biliary coloring is obtained by the positive reaction of Gmelin's test, which is as follows:

We place a specimen of the suspected urine in a test-tube, and with a glass pipette introduce beneath it a layer of concentrated nitric acid to which nitrous acid has been added. The nitric acid should have only a faint yellow color. Chemically pure nitric acid is just as useful as the fuming acid. We obtain an excellent reagent by adding to a larger quantity of pure nitric acid a few drops of the fuming acid, or by heating the pure acid for a short time with a splinter of wood. The continuous oxidation of the biliary coloring matter will then produce at the point of contact of the urine and acid the characteristic *green* zone of biliverdin which, upon allowing the test-tube to stand, is gradually disseminated upward and is subsequently followed by a ring of blue, violet, red and yellow, which slowly rises.

Even with a great dilution of the urine, these bands of color may be distinctly seen if the test-tube is held against a moist piece of filter paper or a plate of ground glass and examined in a clear light.

If we put a drop of nitric acid upon the filter paper through which the urine has been filtered, the characteristic colors will develop in concentric rings. By adding chloroform to some of the urine in a test-tube a yellow color is produced. We may also obtain the Gmelin reaction with chloroform extract by adding commercial nitric acid or bromin water.

The method proposed by Maréchal and Rosin, of covering the urine with a dilute tincture of iodin, will also produce at the point of contact a play of greenish colors which denotes the presence of biliary coloring matter.

Besides biliary coloring matter other constituents of the bile are certainly contained in the urine, chiefly *bile acids.* Complicated chemical methods are necessary to isolate these by Pettenkofer's test.

The urinary sediment obtained by centrifugation shows under the microscope *hyaline casts* which, in the case to be described, were stained a distinct yellow. The presence of albumin in this urine could not be demonstrated by ordinary reagents, nor was sugar present.

The overflooding of the organism with biliary coloring matter is betrayed also by the *intense yellow staining of the blood serum,* which may be recognized in a thin layer of the blood in a glass capillary pipette, particularly on comparison with normal blood serum. In the patient under observation the blood was obtained by means of a capillary pipette after pricking the lobe of the ear, and a Gruber-Widal agglutination test was made with serum which was negative in typhoid cultures as well as in those of paratyphoid.

The serum may also be tested by Gmelin's method. With nitric acid, characteristic colored rings will be observed at the point of contact with the precipitated albumin.

In the case under consideration this accumulation of biliary coloring matter in the organism was coincident with the absence of bile from the intestine. The peculiar, whitish gray color and clayish consistency of the feces are often noticed by the patient.

Yet such appearance in normal feces is not proof of the absence of biliary coloring matter or its reduction products, hydrobilirubin and urobilin. On the contrary, it is demonstrable that the grayish white color is due to a large quantity of fats and soaps in the dejecta. Under the microscope we may discern many fat globules and long fatty acid crystals, also a large number of fine needles of sodium, calcium and magnesium soaps. These soaps give to the feces their peculiar *glistening appearance.*

If the feces are rendered acid by an addition of acetic acid, and are then shaken up with alcohol and ether, many of these colorless masses

may be dissolved, after which the residue shows a dark brown discoloration due to the undigested remains of meat. Neither bilirubin nor urobilin can be demonstrated in the pale yellow ethereal extract, and, in fact, an occlusion of bile from the intestine may be assumed.

The abnormally large amount of fat in the feces is unquestionably the consequence of absence of bile from the intestine, although it is possible that an obstruction to the flow of the pancreatic juice may be simultaneously operative.

The *constipation and flatulence* of which the patients complain must be referred to an inhibition to the flow of bile. Primarily, the normal stimulation of intestinal peristalsis by the bile is lacking.

Whether the absence of the inhibitive effect of bile upon decomposition may be regarded as the cause of the marked production of gas and the somewhat unusual odor of the feces is questionable. Bidder and Schmidt have assumed that the bile exerts an influence upon intestinal decomposition. Nevertheless there can be no doubt that the bile itself may readily decompose, and it certainly exerts no powerful antiseptic effect. Yet we may reasonably suppose that the absence of bile from the intestine favors the development of certain varieties of bacteria, and thus diverts the processes of decomposition into definite tracts. It may be that the alteration in the composition of the intestinal contents from the lack of bile, for instance, the greater proportion of fat, is the sole cause of the abnormal processes of decomposition in the intestine.

The probability that increased decomposition may occasionally result from biliary occlusion is favored by the increased excretion of ethyl sulphuric acid in the urine which has been noted by various authors (Brieger, Biernacki, Eiger, Schmidt, Böhm). But this is apparently not an invariable finding, since other investigators, such as Röhmann, Pott, and v. Noorden, found no marked variation from the normal in the ethyl sulphuric acid contents of the urine in jaundice.

The symptom-complex of jaundice is met with in very unlike but more or less serious affections: In occlusion of the biliary passages from gallstones, in neoplasms of the biliary channels and in the head of the pancreas, in various cases of diffused and circumscribed disease of the liver, especially in certain forms of hepatic cirrhosis. Icteroid discoloration of the skin and mucous membranes of less intensity is observed in other affections, in general circulatory disturbances, in those produced by disease of the heart, in some cases of pneumonia, in general sepsis, and in other infectious diseases. In all of these cases jaundice appears only as an accompanying symptom in contradistinction to the typical symptoms of the disease by which we determine the character of the affection and its probable course.

The circumstances are different in the case under consideration. Here markedly developed icterus dominates the entire clinical picture, and forms

the salient feature of the pathologic condition. Here we are dealing with *simple essential jaundice,* with *icterus simplex.*

This diagnosis corresponds with what we glean from the history of the following case: The patient, a strong man, 26 years of age, and previously healthy, states that about fourteen days ago upon the occasion of a festivity he suffered from a stomach disturbance. The symptoms which appeared within the next few days did not, as on similar occasions, soon pass away, but continued; a sense of discomfort in the gastric region, a disagreeable taste, loss of appetite, a tendency to nausea, as well as general malaise, lassitude, psychical depression, headache, and mild vertigo persisted. At first there was some fever, also slight diarrhea followed by constipation, but no colicky pain. After about a week the yellowness of the patient became noticeable to those about him. Subsequently he remembered that his urine, even for a few days before, was extremely dark and had stained his linen yellow. Until the present time the patient has been able to follow his occupation, but has felt very weak and has emaciated decidedly. Solid food, particularly fatty food, has been repugnant to him, but he has had increased thirst and a special desire for cool and acid drinks.

Further examination revealed no marked change except the jaundice; a few scratches upon the skin showed that the patient was annoyed by itching. There was no elevation of the temperature, which was, on the contrary, somewhat subnormal, 97°–98.1° F.; the pulse was slow, 64 to 68. There was nothing abnormal in the thoracic organs. The abdomen was slightly distended, the hepatic region somewhat sensitive to pressure. The liver showed some enlargement just below the border of the ribs, but was not particularly hard to the touch. The gall-bladder could be distinctly palpated below the border of the liver, and apparently was slightly distended. Splenic dulness seemed somewhat increased, but the spleen could not be palpated.

We therefore recognized the clinical condition known as "*catarrhal*" jaundice, which in typical cases, as a rule, runs a benign course.

We have refrained from the use of this designation because, in its conception, a definite mode of development of the jaundice has been assumed, and this has by no means been proven in all cases. The term is based on the assumption that the jaundice is caused by a catarrhal inflammation of the mucous membrane of the gall-ducts, or of the duodenum at the mouth of the gall-ducts, which *mechanically inhibits the outflow of bile.* The finding of a plug of colorless mucus at the mouth of the common bile duct and the difficulty of forcing the contents of the gall-bladder into the intestine by pressure have, since the time of Virchow, been regarded as the anatomical foundation for this view.

To-day the plug of mucus is no longer considered to be the actual obstruction to the flow of bile. From the evident lack of biliary imbibition

which the mucus reveals, we conclude that, at most, there was an actual impediment to the flow of bile during life. The mechanical obstruction is revealed by the inspissation of the bile itself as well as by the catarrhal swelling of the mucous membrane in the narrow biliary passages. It is true that little of this swelling is to be seen at the autopsy, but the observations of those who have inspected the mucous membranes during life have taught us that swelling, due to marked hyperemia, is not recognizable after death.

Nevertheless, this assumption does not sufficiently explain the development of jaundice in all of the cases which are included under the term catarrhal jaundice; at least, it does not coincide with our ideas of catarrh of the biliary passages, of *cholangitis catarrhalis*. The symptoms of such a cholangitis are often found in cases in which icterus was never present, and are frequently absent when the clinical picture exactly corresponds to essential jaundice.

It is true there are few opportunities for the anatomical investigation of typical cases of this affection, since, as a rule, their course is benign. But their purely clinical investigation, the nature of their appearance, their frequent occurrence under certain conditions, their relation to other forms of jaundice and to diseases of the liver and biliary passages, taken in connection with the results of experimental, physiologic and pathologic investigation, have recently led to other hypotheses which, although by no means clear, are in many respects far in advance of earlier conceptions.

This change of view not only embraces the nosologic position of this special form of jaundice but also explains the processes brought about in these cases by the transference of biliary constituents into the blood and the fluids of the tissues.

ORIGIN

Before discussing further the subject of jaundice, we must express our present opinions in regard to the development of jaundice in general.

At this time we may maintain that all jaundice is of hepatogenous origin, i. e., that general jaundice cannot occur without an implication of the liver. Every case of jaundice, therefore, is due to *absorption of bile which is formed in the liver.*

The old teaching of *"obstructive jaundice,"* the development of jaundice by an inhibited excretion of coloring matter formed outside of the liver and which can be regarded only as an excretion product, has been completely disproven by the investigations of Stern, Naunyn and Minkowski. But the opinion also that the icterus in *"hematogenous"* jaundice is *"anhepatogenous"* is no longer tenable. There can be no doubt that hematoidin (or bilirubin) may be formed from hemoglobin outside the liver, for example, in extravasations of blood. It is also certain that processes which run their course with a massive dissolution of red blood-

corpuscles and an increased destruction of hemoglobin may lead to jaundice, but in such cases, as in hemoglobinemia after poisoning with arseniureted hydrogen, toluilendiamin and other poisons, the transformation of hemoglobin into biliary coloring matter takes place in the liver (Stadelmann, Affanassiew), and its presence there causes the outbreak of jaundice (Naunyn and Minkowski). The latest observations of Ioannovics do not alter the fact that by hemolysis the spleen plays an especial rôle in the production of jaundice, since this organ is actively implicated in the destruction of damaged blood-corpuscles. The designation of such forms of jaundice as "hematogenous" or "hematohepatogenous" is even less justifiable, as the material for the production of biliary coloring matter is normally furnished by the hemoglobin. If we desire to indicate the special mode of development of this form of jaundice, the designations "eythemolytic" and "hemolytic" are certainly preferable.

The most likely cause for the absorption of bile in the liver is a mechanical impediment to the flow of bile in the biliary passages. Therefore, in all cases of jaundice an attempt should first be made to ascertain the nature of this mechanical obstruction. Naturally it is easy to assume a "*stasis icterus*" when either the lumen of one of the large biliary passages is occluded or there is external compression of the biliary passages. When, however, the impermeability of the excretory passages cannot be certainly demonstrated, more or less hypothetical explanations of the obstruction to the flow of bile may be suggested. The fact has been pointed out that the secretory pressure of the bile is comparatively low (according to Heidenhain in the dog about 200 mm., according to Bürker in the rabbit 75 to 80 mm.); therefore even a slight obstruction to the flow of bile is sufficient to cause the absorption of biliary constituents by the blood. Hence it was believed, as has been stated, that the development of simple jaundice could be referred to catarrhal swelling of the mucous membrane and to the accumulation of mucus at the mouth of the common bile duct. Jaundice in infectious diseases was attributed to complicating intestinal catarrh and was also regarded as "catarrhal." When jaundice occurred in pneumonia it was assumed that, in consequence of a decrease in the respiratory excursus of the diaphragm, an important adjunct for the propulsion of bile was absent. Janndice in cardiac disease was referred to a decrease of blood pressure, or to compression of the finer biliary channels by the dilated blood capillaries. In the case of icterus neonatorum, the condition was thought to be due to the altered circulatory relations after birth, to a patulous ductus arantii, and the like. Hepatic cirrhosis was at one time attributed to a compression of the finest interlobular biliary channels by constricting connective tissue, at another time to a catarrhal affection of the finest biliary passages or the occlusion of their lumen by granular pigmented masses, by desquamated and swollen epithelium and the like. Some authors sought for the explanation of the obstruction

to the flow of bile in the morphologic changes, the swelling and displacement of the hepatic cells, and the "dislocation of the hepatic cell trabeculæ."

"Hemolytic" jaundice from the effect of blood poisoning was first referred by Stadelmann to an inspissation of the bile caused by the increased destruction of blood-corpuscles in the liver, which thus formed coloring matter. Naunyn and Minkowski therefore employed for this form of jaundice the designation "*jaundice from polycholia,*" and Stadelmann proposed as a more concise expression, "*pleiochromia.*" It did not escape the attention of these authors that the increased formation of coloring matter was not the only cause of the obstruction to the outflow of bile, but, as Stadelmann emphasized, besides an increased secretion of mucus, the occlusion of the biliary capillaries with decomposed epithelia and hepatic cells destroyed by the poison might also impede the outflow of bile, as well as, perhaps, the compression of the finer biliary passages by swollen glandular cells or dilated blood capillaries which had been subjected to toxic influence.

Although a conception of this kind relegated "polycholic" jaundice to the ranks of jaundice due to mechanical obstruction, many authors have regarded "*polycholia*" as a welcome adjunct by which to explain the different varieties of jaundice whose development from biliary stasis could otherwise scarcely be proven. Above all, icterus neonatorum was ascribed to a suddenly increased formation of biliary coloring material brought about in the first few days after birth (Hofmeier, Silbermann, Hayem) by a massive destruction of the red blood-corpuscles. The development of jaundice from polycholia was also assumed by many authors (Rosenstein, Chauffard, Girode, Banti and others) in certain forms of hepatic cirrhosis as well as in the different varieties of "infectious" jaundice. This was based upon the observation that in such cases, notwithstanding the jaundice, there is usually an intense biliary staining of the feces. Grawitz found that in chronic circulatory disturbances free hemoglobin was demonstrable in the blood serum; he therefore believed that jaundice in cardiac disease might be attributed to polycholia. G. Hoppe-Seyler declared icterus in pneumonia after injections of tuberculin and in Graves' disease to be polycholic, because in these conditions he demonstrated a simultaneous increase of urobilin in the urine and in the feces.

In those forms of icterus in which there could be no doubt of a mechanical obstruction to the flow of bile, some authors went so far as to ascribe the jaundice to the polycholia which caused a destruction of red blood-corpuscles. Browicz, to whom we are indebted for very valuable investigations into the histology of the liver, attempted to prove that a polycholia of this kind was the foundation of *all* forms of jaundice; even in occlusion of the common bile duct he believed that only a mediate influence upon the development of jaundice could be ascribed to the mechanical factor. These

theories, however, did not prove satisfactory. On the one hand, the purely mechanical explanations were incomplete. In some cases the intensity of the jaundice was entirely out of proportion to the demonstrable obstruction; in many others, no obstruction of this kind could be determined, and, finally, in the liver itself signs of biliary stasis were frequently absent. When these signs were present there was undoubtedly a mechanical obstruction to the flow of bile: Dilatation and flooding of the biliary passages, the accumulation of bile in the intralobular bile capillaries, and the deposit of biliary pigment in the hepatic cells. On the other hand, the mere assumption of polycholia or pleiochromia did not sufficiently explain the entrance of bile into the blood and into the fluids of the tissues, unless it were assumed that occasionally the liver ceased to excrete bile normally, while at the same time bile formation was increased.

Thus it came to pass in the last few years that a new view received recognition and a new factor entered into the pathogenesis of icterus; for an attempt was made to attribute the appearance of biliary constituents in the blood to *special functional disturbances of the hepatic cells.*

I was probably the first definitely to express this thought, for in a discussion at the Congress of Internal Medicine in 1892 I made the following remarks: "The special function of the hepatic cell, which enables it to introduce certain products into the bile channels, others into the blood-vessels or lymph channels—therefore biliary coloring matter and bile acids into the biliary passages, and sugar and urea into the blood—is apparently dependent upon the normal nutrition and normal function of the cell itself. Disturbances of this function may exist without mechanical hindrance to the outflow of bile, and may result in the transmission of biliary constituents to the blood." I compared the disturbance in function of the hepatic cells here described with the disturbance in function of the renal cells which occurs in albuminuria, and pointed to the analogous appearance of these forms of disturbance in infectious diseases, in intoxications, in lesions of the nervous system, in circulatory disturbances, and in parenchymatous implication of the organs.

Liebermeister followed a similar train of thought when, a year later, he described "acathectic" jaundice, which was said to be due either to a marked change in the liver cells or because in the process of destruction they could no longer retain the bile or hinder its diffusion into the blood and lymph. Later E. Piek voiced the same opinion when he declared most varieties of jaundice to be essentially due to "paracholia," a disturbance of the flow of bile in a much more general form. For the cases under consideration I proposed the designation "*parapedesis of bile*" in differentiating the close analogy with hemorrhage per rhexin and per diapedesin, also jaundice "per stasin" and "per parapedesin."

Absolute proof of this origin of jaundice is extremely difficult to obtain. But this hypothesis offered not only a plausible interpretation of

those forms of jaundice in particular for which former explanations were unsatisfactory, but it coincided with many other common pathologic hypotheses. It was unnecessary to combine mysterious "vitalistic" ideas with the theory of such a "special functional disturbance" of the hepatic cells as might lead to the excretion of their products of secretion in abnormal directions. We know that nutritive disturbances and pathologic changes of the cells not only modify the physical and chemical processes which occur in glandular secretion, but the function of the cells as "semipermeable membranes" may be so altered as to permit the passage of molecules which cannot pass through the normal cell. And particularly in those varieties of jaundice in which this explanation seemed necessary—jaundice in infectious diseases and intoxications, in circulatory conditions and in disturbances of innervation—such a damage to the function of the cell might, doubtless, readily be supposed. Added to this, the histologic researches of Nauwerck and Fraser, Browicz, and Szubinski afforded an anatomical basis for the assumption of an irregular, intracellular exchange of the different products of the hepatic function.

Thus the teaching of parapedesis of bile soon found general acceptance. Some authors went so far as to emphasize the disturbed function of the liver in *all* forms of jaundice, and attached little importance to the mechanical factor in biliary stasis, particularly in the jaundice occurring in cholelithiasis which was formerly regarded as the type of obstructive jaundice.

Conflicting opinions were not lacking. First, D. Gerhardt opposed the view that in the genesis of simple stasis icterus the main point was the abnormal direction of the cell secretion. Subsequently Hans Eppinger attempted to overthrow the entire principle of parapedesis of bile because, by painstaking anatomical investigations, he determined the factor of biliary stasis to be of much greater significance.

Eppinger devised a method of staining by means of which all the processes of the interacinous biliary capillaries were made distinctly visible, and he employed this method not only to determine the normal course of these biliary capillaries between the hepatic cells and their entrance into the liver cells, even in the human liver, but also to test the action of the biliary capillaries in the different forms of jaundice. He observed first that in pure mechanical stasis icterus—after tying the hepatic duct—the accumulation of bile in the biliary passages led to dilatation, tortuosity, lengthening and varicose distention of the intracellular biliary passages until they finally ruptured and their contents submerged the perivascular lymph spaces. Later he found the same dilatation and rupture of the interacinous biliary capillaries in those cases in which jaundice was due to hepatic cirrhosis, poisoning from phosphorus, or from circulatory disturbances. He believed, therefore, that these cases could only be regarded as special forms of mechanical stasis icterus. The obstruction to the

outflow of bile was, however, not in the large bile ducts but in their finest branches, which, as in the case of hepatic cirrhosis, either were compressed by proliferating connective tissue or, as in the case of toxic and cyanotic jaundice, by coagulated masses of bile, by *"biliary thrombi."*

Investigations similar to those of Eppinger have recently been made by Abramow and Samoilowicz, who, in the main, confirm the former's results. These authors also assume, as an absolute prerequisite for the development of jaundice, a free flow of bile in the perivascular lymph spaces.

Elsewhere I recently criticized Eppinger's views, and especially emphasized the fact that, in judging such conditions, the interchange of material occurring in the organism could by no means be gauged by morphologic observations alone. The appearance of the previously mentioned biliary thrombi proves that the secretion of the liver is no longer of normal composition, and that abnormal processes of secretion have already taken place in the hepatic cells. We know that in certain maladies, especially in infectious diseases of the liver, the bile may contain albumin. But in these cases especially, notwithstanding an existing jaundice, we frequently fail to find in the liver any signs of biliary stasis, even upon minute microscopic examination. Deposits of "coagulated bile," of precipitates of biliary coloring matter in the intracellular biliary capillaries and within the hepatic cells themselves, are not found in cases of recent jaundice but only when this has existed for considerable time. They are not the cause of jaundice, but the consequence of functional disturbance of the hepatic cells leading to jaundice. Biliary thrombi are, at most, to be regarded as predisposing factors for the transference of biliary constituents into the blood or the lymph, just as the occlusion of the uriniferous tubules by urinary casts is regarded merely as a predisposing factor for a functional disturbance of the renal capillaries which usually causes the retention of urinary substances.

For the present, notwithstanding these objections, I believe it justifiable to maintain the importance of an abnormal direction of the hepatic secretion in the transference of biliary constituents into the blood, and I therefore briefly define the pathogenesis of jaundice as follows:

General jaundice develops only from the absorption of bile within the liver.

The absorption of bile may be due to the following causes:

1. *An obstruction of the biliary passages which inhibits the outflow of bile (stasis icterus, icterus per stasin).*

2. *A disturbance in the function of the hepatic cells which results in the excretion of biliary constituents in an abnormal direction (diffusion icterus, icterus per parapedesin).*

Naturally no sharp differentiation of these forms of jaundice according to their mode of development can be made, since both factors may be, and

usually are, combined: Mechanical stasis of bile leads to injury of the hepatic cells which results in a disturbance of their secretory function. And any damage of the hepatic cells may be followed by a mechanical obstruction to the flow of bile, either because the lesion may at the same time affect the mucous membrane of the intestine or of the biliary passages, and hence obstruct the flow of bile, or because from the pathologically altered consistence of the bile its outflow becomes difficult, or because the finer biliary passages are occluded by a swelling of the hepatic cells, by dislocation of the hepatic cell trabeculæ, by connective tissue proliferation, or by biliary thrombi.

In the incipient stages, as a rule, one or the other factor preponderates, and promotes the development of jaundice:

Mechanical obstruction comes into question in the following conditions:

(a) In *occlusion of the lumen of the biliary channels* by *gall-stones* or *foreign bodies* entering from the intestine (fruit kernels, thread worms, distoma, and the like), by *neoplasms* and *cicatricial strictures*;

(b) In *external compression of the biliary channels by tumors* which originate in the stomach, the intestine, the head of the pancreas, the peritoneum, the portal lymph-glands, in the gall-bladder or the liver itself; by *perihepatic bands,* by *floating kidneys,* by *aneurysms of the abdominal aorta,* of the celiac axis, the hepatic artery or the superior mesenteric artery, in rare cases even by *tumors of the uterus and ovaries,* or by the impaction of *fecal masses* in the right flexure of the colon.

Functional disturbances of the hepatic cells promote the development of jaundice:

(a) In *a disturbance of the circulation of the blood* in the liver due to *cardiac disease, thrombosis of the portal vein,* etc.;

(b) In disturbances of innervation of the liver, as, for example, in icterus following psychical emotion, *icterus ex emotione*;

(c) From the effect of *poisons* (phosphorus, arseniureted hydrogen, and others) and *toxic bacterial products* introduced with tainted food (*ptomains*) or those formed in the organism in infectious diseases (*toxins*), in pneumonia, septicemia, relapsing fever, bilious typhoid, yellow fever, syphilis, and the like.

SYMPTOMS

We have seen that the most diverse constitutional diseases and the most dissimilar affections of the liver and other organs may lead, in one or the other of the modes here described, or in both simultaneously, to the symptom-complex of jaundice. The importance of the jaundice in the conception of the pathologic condition of the individual case varies, according to whether this symptom-complex is distinctly developed or less so, and whether the symptoms that accompany it—which at one time may be due to the systemic disease, at other times to a change in the liver

or to a simultaneous affection of other organs—become more or less prominent in the entire clinical picture. In many cases we regard jaundice only as a *symptom* or a *complication* of another pathologic condition. In others, however, **jaundice assumes the importance of a substantive affection.**

It is not always possible to draw a sharp line of demarcation between this substantive form of jaundice and the jaundice which occurs in organic disease of the liver or the infectious processes or intoxications which lead to jaundice.

The conditions are nowise different from those of other pathologic changes produced by the effect of various deleterious agents in the organism. Whether, for example, in the individual case, we speak of septic nephritis, myocarditis, and the like, or of sepsis with albuminuria and cardiac insufficiency, depends only upon the prominent implication of individual parts of the organism in comparison with the general symptoms.

From this point of view we can readily understand that the numerous cases of simple jaundice, no matter how characteristic the mode of their appearance and their development, can by no means always be regarded as of the same nature. And, on the other hand, it immediately becomes obvious that in simple jaundice, from the benign to the most severe forms, those with decided constitutional affection and very marked changes in the liver, icterus gravis and icterus infectiosus, there is every possible transitional stage. In one instance various factors may act conjointly upon the liver and the biliary passages so that the symptoms of simple jaundice are produced. In another, the same cause may generate effects of varying intensity so that at one time serious, at other times mild, affections result.

The milder cases of simple jaundice, as already stated, are included under the conception of *"catarrhal" jaundice.* In fact, *catarrhal cholangitis* appears to be the primary and principal anatomical foundation of the pathologic picture which has been described. By an inflammation of the mucous membrane of the biliary channels and an increased excretion of mucus this cholangitis may obstruct the flow of bile, and thus mechanically lead to stasis icterus.[1]

Cholangitis, like all inflammations of the mucous membrane, may be produced by *toxic* or *infectious* processes of any kind. Deleterious products may enter the bile *from the blood,* may pass through the liver into the biliary passages, or enter into the biliary channels from the intestine. In the case first mentioned, *secondary infection* may play a great rôle by the entrance of bacteria from the intestine.

But, as has been stated, the theory that jaundice originates from the

[1] In a case of catarrhal jaundice, Kimura estimated the viscosity of the bile at 58.24 in comparison with a normal viscosity of 13.21.

mechanical effects of a catarrh of the mucous membrane has shown itself to be unsatisfactory. Here, too, we must allude to the possibility that cholangitis may be the cause as well as the consequence of biliary stasis.

It is true we know that bile is sometimes neither aseptic nor sterile, but, under normal circumstances, it certainly contains but few bacteria and these are non-pathogenic. The narrow mouth of the common bile duct, which is frequently washed by the flow of bile, prevents the ingress and development of microörganisms from the intestine.

As soon as the outflow of bile is impeded, the bile duct may be invaded from the intestine by the most varied pathogenic microbes: The bacterium coli, the different varieties of proteus, staphylococci, streptococci, pneumococci, typhoid bacilli and others, penetrate into the bile channels and there find in the "residual bile" a suitable soil for their development. *Thus any check to the outflow of bile may lead to infection of the biliary passages and inflammatory changes in its mucous membrane.*

Thus all mechanical obstructions of the biliary passages, whether occlusion of the lumen of the bile ducts or their compression from without, favor the production of cholangitis. But diseases of the liver which lead to a disturbance of the nutrition and function of the hepatic cells may also obstruct the flow of bile, and facilitate the development of cholangitis. As a matter of fact we frequently find *cholangitis to be a complication of the most varied acute and chronic forms of disease of the liver and bile channels.*

This complicating cholangitis increases the hindrance to the propulsion of bile and promotes the development of jaundice in the diseases we have enumerated, hence jaundice in gall-stones, in hepatic cirrhosis, and the like, has usually been regarded as "inflammatory," and has been attributed to a complication with cholangitis.

Of late, it has frequently been pointed out that these noxious effects, toxic and infectious influences, which we consider the primal cause of cholangitis, not only affect the mucous membrane of the intestine and the bile channels but also the parenchyma of the liver, and, therefore, even *in simple jaundice, an affection of the hepatic parenchyma and disturbance of the hepatic cell function* have been held responsible for the appearance of the clinical symptoms.

Some authors, among them Chauffard, have maintained that the action of *poisons* which are introduced with the food or which develop within the digestive tract by abnormal fermentation and decomposition, is conveyed to the liver by the portal vein. Others, such as Gilbert and Girode, Charrin and Roger, attach the greatest significance to the *local infection* of the liver by the ingress of bacteria from the intestine. Still others, such as Botkin, Kelsch, and Heitler, in these cases believe only in a *general* infection which, by its special localization in the liver or by the action

of the toxins which the organism develops in the liver, brings about an obstruction to the flow of bile.

This last conception found a firm support in the observation of cases of jaundice, which are on the increase, in which the action of the infectious agent betrayed itself simultaneously in the *clinical course of the individual case* and in the *epidemic appearance of kindred affections.*

Simple jaundice, as we may perceive from the history previously quoted, is not rarely accompanied by more or less well developed *symptoms of a general infection*—fever, enlargement of the spleen, albuminuria. In these cases we speak of *"infectious jaundice."* In the mildest cases these symptoms of general infection are scarcely perceptible or only temporarily so; often they appear only in the first days of the disease, and are readily overlooked. In some cases, however, they become much more noticeable as the disease progresses, and, finally, there are cases which run their course with the most severe symptoms of a general febrile affection, cases in which jaundice is merely a concomitant phenomenon of the entire pathologic condition.

Such types, the severe as well as the mild, have occasionally appeared in epidemics. This *"epidemic jaundice"* has frequently been regarded as a *special specific infectious disease,* and many authors have attempted to explain *isolated cases of simple jaundice occurring at the same time as "sporadic cases" of this infectious disease.*

Numerous observations of such epidemics, appearing under most varying circumstances, have been reported. These observations, however, by no means confirm the view that the disease is invariably due to a definite *specific* pathogenic agent. It is true that for the development of the different cases occurring in a single epidemic one and the same pathogenic agent might be assumed to be the cause, as, for instance, in those epidemics which Lürmann observed in Bremen occurring after re-vaccination, or those which Rizet observed in soldiers engaged in cleaning the moat of a fortress, etc. But it is by no means proven, or even likely, that in these epidemics the same pathogenic agent was always operative. Not only have the bacteriologic investigations led to a different conclusion, but all which has been learned regarding the cause, the manner of distribution, the incubation of the disease in the various epidemics, is opposed to the view that the same specific pathogenic agent was the cause.

Even the attempt to differentiate from the numerous cases of infectious jaundice a definite group characterized by a peculiar course of the disease as an especial infectious disease, the so-called **"Weil's disease,"** has so far been unsuccessful.[1]

In the year 1886 Weil described the first four cases of a peculiar disease running its course "with splenic tumor, jaundice and nephritis," which

[1] See volume "Infectious Diseases," page 808.

was characterized by a special febrile course with a tendency to relapses; this affection was subsequently named after him. Since that time numerous publications have appeared concerning the disease; in 1901 Fränkel collected from literature no less than 150 cases. Nevertheless, even to-day, the views regarding the nature of this symptom-complex are wide asunder. Some authors, such as Fiedler, Neumann, and Münzer, share Weil's opinion that it is a special infectious disease due to a specific pathogenic agent. Many others, such as Brosch, Cramer, Stirl, Pfuhl, Fränkel, Nauwerck, Karlinsky, and Soupault, assume that the same pathologic picture is produced by various causes, and that the cases described as Weil's disease have masked various infectious diseases and intoxications: Enteric fever and relapsing fever, infectious intestinal catarrh, tubercular pericarditis, santonin and ptomain poisoning and the like, and many others which have run their course with phenomena resembling Weil's symptom-complex.

During an epidemic of infectious jaundice in the year 1892 Jäger isolated a special microörganism, the pleomorphic *bacillus proteus fluorescens,* which he regarded as the pathogenic agent. The specificity of this bacillus was doubted. Yet it appears to be a fact that this variety of proteus is capable of producing jaundice, although it has as yet by no means been proven that a definite characteristic course of the disease may be attributed to the action of this special bacillus.

While it is perhaps true that many isolated cases of simple jaundice may be referred to the same origin as cases of epidemic jaundice—nevertheless, the former need not be regarded as sporadic cases of a well marked infectious disease, but, on the contrary, even in these a very different etiology may be assigned for the jaundice.[1]

Naturally these deleterious agents frequently enter the stomach and intestinal canal with the food, and develop within the digestive tract under the influence of abnormal processes of decomposition which the ingesta there undergo. This enables us to understand why, as a rule, the symptoms of gastric and intestinal catarrh introduce the clinical picture, and why the development of jaundice is usually referred to the same etiologic factors which we are accustomed to regard as the causes of gastric and intestinal catarrhs: *The immoderate consumption of food, the ingestion of food difficult to digest or that is tainted, of food too cold or too hot, the abuse of alcohol, refrigeration, infections and intoxications.*

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[1] From this point of view the recent contradictory reports regarding the *agglutinating properties of the blood serum of patients with jaundice* may possibly be understood. Among the cases of jaundice many are due to an infection with typhoid bacilli or other closely related varieties of bacteria.

HEPATIC INSUFFICIENCY

The action of various deleterious agents upon the liver does not limit itself to a mere disturbance of the excretion of bile. Other functions of the liver also may be impaired to greater or less extent by the same pathologic causes, and **hepatic insufficiency** may be added to the jaundice.

The disturbance of other functions of the liver is not so obvious as the obstruction to the flow of bile which causes jaundice. Numerous physiologic investigations have revealed to us much concerning the multiplicity of the functions of the liver, not only regarding the processes by which the biliary constituents are produced, but also relative to the part which the liver plays in the metabolism of the most important foods and constituents of the body, the carbohydrates, fats, and proteids. Endeavors to investigate the disturbance of these functions by experimental pathologic means have not been lacking. But in diseases of the liver we are as yet not in position to trace these disturbances in individual organs.

As the secretion of the liver is insusceptible to direct investigation except in certain exceptional instances, such as biliary fistula, we are unable to frame a positive opinion as to the *quantitative and qualitative changes in bile production.* Studies of the human gall-bladder, such as were recently published by Tokuye Kimura, only prove primarily that in pathologic lesions there are marked differences in the condition of the bile. No definite idea can be obtained from these investigations in regard to the secretion of bile, for here, among other variations, the secretion of the mucous membrane of the gall-bladder as well as the inspissation and decomposition of the bile in the gall-bladder must be considered.

For the time being we know only that a decrease in the production of bile, a *hypocholia* or *oligocholia,* occurs in various diseases of the liver and, as a rule, also in consequence of prolonged obstruction to the flow of bile, and that an increased production of bile or biliary coloring matter, a *polycholia* or *pleiochromia,* may appear after the addition of biliary constituents or after the effect of poisons which produce dissolution of blood-corpuscles.

Whether the production of urobilin instead of bilirubin and *urobilinuria* may be regarded as the expression of a disturbed function of the liver is still a mooted question. In most cases the urobilin excreted in the urine is of enterogenous origin, and develops from the reduction of bilirubin in intestinal decomposition. In jaundice an increased excretion of urobilin as well as bilirubin is frequently found in the urine; in many cases, or in certain stages, even urobilin exclusively instead of bilirubin. However, according to the investigations of Fr. Müller, even in these cases urobilin is probably formed only in the intestine. According to Kimura even the urobilin, that is urobilinogen (Braunstein), which is almost invariably found in the bile, appears to be of enterogenous origin. In com-

plete occlusion of the bile from the intestine it is absent both from the bile and from the urine.

The view that an accumulation of urobilin in the blood may produce an especial form of icterus, the "*urobilin icterus*" of C. Gerhardt (Gubler's "ictère hémaphéique"), may be regarded as disproven. It has been determined by numerous researches that even in those cases in which the urine contains urobilin alone the yellow color of the skin, of the blood serum, and of the serous effusions, is exclusively due to bilirubin.

As additional consequences of disturbed hepatic function we must consider the *diminished excretion of urea with an increased excretion of ammonia,* so-called *alimentary glycosuria,* as well as an *increased toxicity of the urine.* But these phenomena cannot as yet be utilized in differentiating hepatic insufficiency. On the contrary, the disturbed transformation of other carbohydrates into grape-sugar, which is expressed in *alimentary levulosuria,* has a certain significance as a sign of disturbed function of the liver.

In the *clinical picture* the *milder grades* of hepatic insufficiency manifest at most only very slight and varying symptoms, such as may appear in any other disease. It is therefore, at present, a purely arbitrary assumption when we designate certain symptoms of dyspepsia which are associated with psychical depression, a tendency to headache, uncomfortable sensations in the abdomen, and more or less distinctly developed jaundice, as "hepatic dyspepsia," "biliousness," "torpor of the liver," and the like, as is commonly done both in England and America.

But there can be no doubt that certain grave symptoms which we meet with when an especially severe damage to the liver has taken place are chiefly to be attributed to an absence of hepatic function—to *acholia,* as the condition was named by Frerichs, with particular reference to the formation of bile, or to *hepatargy,* the designation proposed by Quincke.

Usually there are severe *disturbances on the part of the central nervous system,* similar to those observed in the most varied *autointoxications*: Severe *headache, nausea and vomiting, implication of the sensorium* even to most profound *coma,* a state of *mental irritability* and *delirium,* even *maniacal attacks, muscular twitchings,* and *general convulsions.* To these must be added the development of the *hemorrhagic diathesis,* a tendency to hemorrhage from every possible organ, as well as the appearance of *circulatory disturbances,* and finally *collapse* which brings about the *fatal issue.*

When these severe symptoms are superadded to jaundice we have the condition known as **icterus gravis.**

ACUTE YELLOW ATROPHY

The clinical picture of icterus gravis is, as a rule, associated anatomically with an *acute diffuse degeneration of the parenchyma of the liver* which leads to the destruction of the hepatic cells and a rapid decrease in the size of the organ which usually is designated as "*acute yellow atrophy of the liver.*" This designation, however, applies only to the appearance of the liver in the advanced stage of anatomical change which cases running a typical clinical course usually attain. But the intensity of the clinical symptoms by no means invariably corresponds with the intensity of the anatomical changes. Often the patient perishes before the anatomical lesion of this characteristic stage is reached, therefore the term "icterus gravis" does not aptly describe the condition known as "acute yellow atrophy of the liver."

In these cases of fatal jaundice it is difficult to differentiate the disturbances which are due to the absence of hepatic function and those due to the consequent alteration in the composition of the blood, the *hepatotoxemia* or hepatic autointoxication from the toxic effects of biliary constituents which are circulating in the organism, the *cholemia* from those symptoms produced by the primary *general infection* or *general intoxication.*

Until very recently attempts have been constantly made to prove by experimental investigation that these severe symptoms depend mainly upon the action of biliary constituents circulating in the blood, especially of the *bile acid salts.* A few years ago Bickel demonstrated experimentally that the biliary salts when brought directly into contact with the surfaces of the cerebral hemispheres prove themselves to be especially potent poisons to the nervous system.

Nevertheless it is questionable whether the biliary salts ever attain in the organism such a concentration as is necessary in the experiment to produce a toxic action.

With some degree of probability, we may refer to cholemia in a restricted sense, that is, to the *over-flooding of the organism with biliary constituents,* the following:

The *slowing of the pulse* so frequently observed in jaundice, which is partially due to the direct action of the bile acids upon the heart muscle, partially to central stimulation of the inhibitive fibers contained in the pneumogastric nerve; also certain *milder disturbances* in the *nervous and muscular apparatus* which are expressed by itching of the skin, disturbance of sight, particularly xanthopsia, headache, general lassitude, muscular weakness, and psychical depression; furthermore, a *damage to the kidneys* which is chiefly revealed anatomically by certain alterations in the epithelia and in the convoluted uriniferous tubules, and is clinically manifested by the appearance of albuminuria and the excretion of casts which, as Noth-

nagel has shown, may also be found in the urine of jaundice when albumin cannot be demonstrated by the ordinary tests.

Whether the property of the bile acids *to dissolve red blood-corpuscles* is also called into action in jaundice is very questionable on account of the slight concentration of the biliary salts in the organism. Nor is it certain that we can refer to the action of the biliary acids the changes in the walls of the vessels which are the foundation of the *hemorrhagic diathesis,* and which occur in severe cases of jaundice.

On the contrary, in cases of icterus gravis, probably all of the other symptoms of severe autointoxication are chiefly due to a *damage of the hepatic function* and an alteration in the composition of the fluids produced by hepatic insufficiency. For these phenomena are also observed after experimental exclusion of the hepatic function as well as in severe hepatic diseases in which there is no marked jaundice.

The special cause of the toxic effect here produced cannot at present be decided with certainty. Probably, however, the substances whose accumulation in the organism brings about this autointoxication are to be sought for in certain *prior nitrogenous stages of urea* the further development of which occurs in the liver; or in the *toxic products of intestinal decomposition* which, under normal circumstances, are deprived of their deleterious power by passing through the liver; or in the *products of decomposition of the hepatic tissue* itself, which may be destroyed by the action of the same deleterious agent which generates the disease.

SYMPTOMS

If, from the views here expressed, we consider that the difference between benign simple jaundice and that running a fatal course, icterus gravis, is merely a difference in the degree of *intensity* of the action of the same or similar factors upon the same tissue elements, it is immediately obvious that no sharp line can be drawn between these forms of jaundice.

As a rule, these forms are ushered in with the same symptoms—those which we recognized in the patient whose history has been related. Only the subsequent course of the disease varies.

In ordinary cases of *simple jaundice,* the symptoms, as we have seen, last for a short time, generally from two to three weeks, with but slight variation in their intensity, after which recovery gradually sets in. The appetite returns, the patient is more comfortable, the digestive disturbances entirely disappear. The feces begin to show biliary staining, the return of bile to the intestine often being indicated previously by the presence of urobilin in the alcoholic ethereal extract of feces which have been rendered acid. The biliary coloring matter in the urine gradually decreases; often a more decided urobilinuria may transitorily appear. The yellowish

discoloration of the skin and conjunctivæ may last longer than the other symptoms. Finally, this also completely disappears, and in three or four weeks after the onset of the disease the patient entirely recovers.

But there may be deviations from this course. Not rarely, particularly in epidemic jaundice, digestive disturbances and constitutional symptoms are very slight from the onset, or may be absent entirely. Only the ictcroid discoloration of the skin and the urine and the clay-colored stools betray the inhibition to the flow of bile. In such cases the duration of the disease is less, being only from ten to twelve days.

In other and much rarer cases, the disease is very protracted, often lasting three or four months, or even longer. Occasionally, in spite of the prolonged course, the reaction upon the constitutional condition is very slight. As a rule, however, these cases gradually increase in severity, and produce a decided loss in strength, emaciation, and anemia. Convalescence in such cases may be correspondingly protracted. Finally, however, complete recovery occurs. Rosenstein reports a case in which catarrhal jaundice lasted more than a year, but terminated in restoration to health. In such instances, however, the diagnosis must be considered very questionable.

Occasionally these cases become more grave. More or less suddenly, severe symptoms of cholemic intoxication and hepatic insufficiency appear, combined with a failure in renal and cardiac activity, or the patients perish from the addition of the hemorrhagic diathesis. These cases, therefore, are examples of a transitional change into the critical state of *icterus gravis*.

Typical cases of the latter form usually present the picture of benign jaundice only during the first days. From the onset the constitutional symptoms are somewhat more intense, and often enlargement and marked sensitiveness of the liver upon pressure are early noticeable. Splenic tumor and albuminuria may be demonstrated even in the first days of the disease. As a rule, however, these cases at the onset do not differ greatly from those previously described.

But soon, after a few days, toward the end of the first or during the second week, rarely after a longer course, a change ensues: The headache increases and sometimes becomes exceedingly severe, the patient is restless, excited, unable to sleep and begins to be delirious. The delirium increases and occasionally assumes a furibund character. The patient screams and tosses about and can hardly be kept in bed. In other cases the delirium may be of a more quiet nature, or from the onset there is a tendency to apathy and somnolence. Finally the sensorium is more and more benumbed, the patient becomes soporous, and then sinks into the deepest coma. During this time there are convulsions in individual muscle groups, with grinding of the teeth, attacks of trismus, often even general convulsions. Vomiting repeatedly occurs, the vomitus sometimes contain-

ing blood in greater or less amounts. Profuse gastric and intestinal hemorrhages may also appear, and there may be bleeding from other parts of the body in addition: Petechiæ and ecchymoses upon the skin and mucous membranes, severe epistaxis, bleeding from the gums, hemorrhages from the urinary passages, and, exceptionally, also pulmonary hemorrhage.

Meantime, conspicuous changes take place in the liver. The organ which was at first enlarged begins to decrease in size. The extent of this decrease may be recognized by examinations repeated from day to day. Finally, the organ is scarcely, if at all, recognizable upon percussion. The spleen, on the other hand, is usually enlarged.

The excretion of urine is diminished. The urine shows jaundice, contains albumin and casts, but little urea; on the other hand leucin and tyrosin are excreted as sediments, or in testing the urine are readily demonstrated.

During this time the temperature is frequently subnormal; sometimes, however, it may be normal or even show febrile ranges. The pulse, which after the appearance of jaundice frequently becomes slow, subsequently is more rapid, smaller, and weaker. Finally, the pulse rate shows extraordinary rapidity but loss in strength, and it rapidly declines, the respiration becomes irregular and interrupted, pulmonary edema appears, and the patient perishes in profound coma.

The entire course may be so extremely fulminant that even four or five days after the appearance of jaundice, and one to three days after the severe cerebral symptoms have become prominent, death may occur. In a few rare cases a course even more rapid has been observed. A somewhat protracted course is less rare: The initial stage may last for two or three weeks or even longer, and after the appearance of the severe symptoms the duration of life is sometimes from twelve to fifteen days.

In the cases with characteristic symptoms which have just been described a favorable course is extremely rare; but several undoubted instances of this kind have been reported. Recovery is then very slow, and convalescence is exceedingly protracted. Sometimes recovery sets in with the sudden appearance of profuse diuresis ("crise urinaire polyurique," Bouchard) or a profuse diarrhea. Harmful products are thus apparently removed from the body. Gradually the hemorrhage ceases, the mind clears, the general condition improves. As a rule, the liver regains its normal size very slowly, but occasionally this may be with extraordinary rapidity. Many months may pass before the debilitated and extremely emaciated patients completely recover their strength.

Like the mildest cases of jaundice, the symptom-complex of icterus gravis may occur secondarily in the course of various chronic diseases of the liver: as, for instance, in the different forms of chronic diffuse hepatitis, more rarely in congestion of the liver, syphilis of the liver, hepatic carcinoma, and others. In such cases the affection often shows the char-

acteristics of a terminal complication. The disturbances on the part of the nervous system rarely appear suddenly, but, as a rule, insidiously and gradually, and they soon increase to the deepest coma or to delirium and convulsions; hemorrhages are added, characteristic changes in the urine appear, but are less prominent than in the cases of primary disease. There may be no decrease in the size of the liver; the fever may be slight or entirely absent, the temperature is usually subnormal; death results in a few days.

Icterus gravis sometimes occurs apparently as a primary disease, without any previous symptoms denoting disease of the liver, and runs a characteristic course; only the autopsy then reveals an earlier affection of the liver the course of which has been entirely latent.

ETIOLOGY

The *causes* of the disease will decide the character of the pathologic picture, especially the severity of the clinical symptoms in the individual case and, above all, the *intensity of the toxic action or the virulence of the pathogenic agent.* Definite noxious agents are characterized by their special effect upon the liver. Whether they lead only to temporary functional damage of the liver, or to acute, rapidly progressing, destructive processes, or to chronic degeneration and inflammation, depends upon their *mode of introduction* into the organism, and upon the *duration and intensity* of their action.

Thus, for example, *acute phosphorus poisoning* with only slight toxic effect may lead to mild transitory forms of jaundice. Larger quantities of the poison will produce the clinical picture of icterus gravis and develop the characteristic changes of acute yellow atrophy of the liver, unless the other effects upon the organism should cause death before the changes in the liver develop to such an extent. Phosphorus rarely acts so powerfully in man as to cause a chronic diffuse hepatitis, but changes which correspond to hepatic cirrhosis have been experimentally produced in animals by repeated doses of small amounts of phosphorus.

On the other hand, we know that *alcohol,* which, as a rule, has a less intense action upon the liver but more frequently implicates this organ, produces chronic hepatitis. A fatal icterus gravis or acute yellow atrophy of the liver has been only exceptionally observed after acute intoxication with alcohol. After acute alcoholic intoxication followed by gastric and intestinal catarrh, a benign icterus has been seen which was looked upon as "catarrhal," but which, perhaps, was also dependent upon a direct action of the alcohol upon the liver.

Among infectious diseases there are some which particularly implicate the liver so that the jaundice or acute diffuse hepatitis actually forms a part of the clinical picture. In other infections it appears to be only a

temporary complication. In the first group *yellow fever,* above all, must be included, for in this the implication of the liver plays such a rôle that Liebermeister remarked: "Yellow fever is merely an acute parenchymatous degeneration of the liver due to a specific infection, and, aside from the effect of the toxins of the disease upon the liver, there is no reason to assume a direct action of the same in other organs." Comparatively often mild and even severe forms of jaundice occur in *relapsing fever* ("bilious typhoid"), also in *septic* and *pyemic affections,* but less frequently in *enteric fever, cholera, diphtheria, malaria,* and *tuberculosis.*

The occurrence of milder forms of jaundice as well as highly developed cases of acute yellow atrophy in *syphilis* requires especial mention. Where this occurs in the early period of the disease during the time of the eruption of the secondary exanthems, it occasionally appears in a very characteristic manner.

Furthermore, nearly all pathogenic microörganisms may occasionally produce an outbreak of malignant or benign jaundice, so that we have no more reason to regard an "essential" icterus gravis or a "primary" acute yellow atrophy of the liver as a definite specific disease than in the case of simple jaundice.

A predisposition to the severe forms of the disease depends upon the non-resistance of the organism as well as upon the intensity of the effect of the poison. This resistance of the organ may have been diminished by a previously existing pathologic change. We observe, therefore, as stated, that the mild as well as the severe forms of jaundice frequently occur as complications of hepatic cirrhosis, congestion of the liver, hepatic syphilis, etc. Yet any impediment to the flow of bile may damage the liver and diminish its power of resistance to poisons and toxins of any kind. *Therefore every jaundice, no matter what its cause, may in its further course lead to hepatic insufficiency and terminate in icterus gravis.*

As another factor which may cause a special predisposition to jaundice, and particularly to the severe forms, *pregnancy* must be mentioned. It is remarkable that about one-half of all the cases described in literature of "primary" acute yellow atrophy of the liver occurring in women have been in pregnant women. We cannot regard these cases as solely due to the influence of puerperal septicemia since only a few occurred in the puerperium, the majority having been observed between the fourth and seventh months of pregnancy.

The special predisposition of the pregnant woman to severe attacks of jaundice is revealed by the fact that, in different epidemics of jaundice, even in those which were largely of benign character, among those who perished with the symptoms of icterus gravis the number of pregnant women was conspicuously large. The French authors (Saint-Vel, Carpentier), in particular, have described such epidemics.

The cause of this extreme susceptibility of the pregnant cannot be

found in the mechanical effect of the gravid uterus, as Klebs has supposed, since the disease occurs even during the earliest months of pregnancy. It is probably the consequence of the changes which the hepatic cells as well as the renal epithelium and other tissue elements undergo during the course of pregnancy.

Brauer, who recently reported a peculiar case in which jaundice recurred during four successive pregnancies, refers the damage of the liver to the preceding action of placental toxins (Veit's syncytiolysins), connecting them with the kidney of pregnancy, with eclampsia, osteomalacia, and other expressions of gravid toxicoses. Brauer attempts to explain *menstrual* jaundice also in a similar manner.

That a *congenital,* often *hereditary, predisposition* may bring about a lessened resistance of the liver and a tendency to mild or severe forms of jaundice is extremely likely. Such a hereditary predisposition to chronic icterus occurring in families is shown in a remarkable manner by certain cases to which I shall briefly refer:

Cases of chronic jaundice appearing in several members of the same family have been described by earlier authors (Murchison). Recent publications are also at hand (Minkowski, Gilbert et Lerebouillet, A. Pick and others). Under the name of "hereditary jaundice" or of "cholémie familiale" very different conditions have been portrayed, and the French authors especially, with their tendency to "diatheses," have attached great significance to the most heterogenous phenomena as expressing hereditary predisposition depending upon family cholemia. In some cases the cause of hereditary jaundice has been sought in a congenital and hereditary constriction of the biliary passages. Pick assumes for his cases either a congenital communication between the lymph channels and biliary passages or a congenital insufficiency of the hepatic cells.

To me it appears questionable whether in all cases of hereditary jaundice a primary change in the liver or in the biliary passages must be assumed. I do not believe this to have occurred in the cases I described as a "peculiar hereditary affection running its course under the picture of chronic jaundice with urobilinuria, splenomegalia and renal siderosis" (*Verhandlungen des XVIII. Congresses für innere Medicin,* 1900).

This symptom-complex occurred in at least eight members of a family in three generations without producing any serious disturbance of the constitutional condition. In one case, upon which an autopsy was held, death being due to other cause, noteworthy structural changes could not be determined either in the liver or in the biliary channels; however, in addition to the marked hyperplasia of the spleen, a very conspicuous deposit of iron was found in the kidneys, which, so far as I am aware, has not been observed in any other condition. At that time, therefore, I expressed the belief that this was due to an especial, transmissible, hereditary anomaly in the metabolism of blood pigment which only seconda-

rily led to disturbance of the function of the liver. During the discussion, Senator reported similar observations and, in the main, agreed with me.

In their clinical course the cases resembled the clinical picture described by Hayem and Lévy as "ictère chronique infectieux splénomégalique," which Gilbert and Lerebouillet have also designated as "cholémie familiale." But the mere fact of the hereditary appearance in successive generations is against Hayem's conception of jaundice as an infectious disease, a conception which was also opposed by Bettmann who described a similar case.

Bettmann determined periodically in his case a hemoglobinemia without hemoglobinuria, and therefore considers a congenital abnormality of the blood to be the basis of the disease.

Recently I met another family which shows characteristically the affection I have described. One member of this family, then a student of medicine, was examined by me twelve years ago. On account of jaundice, urobilinuria, and an enormous splenic tumor, I diagnosticated hepatic cirrhosis, nothing militating against such diagnosis except the absence of an etiologic factor, and, since that time, the continuously undisturbed general condition. After the appearance of my publication this gentleman, who has since become a physician, brought to me his brother, who since childhood has also shown ictcroid discoloration of the skin, has voided a dark urine rich in urobilin, and has also had a large splenic tumor. The father of the patient is said to have died at an advanced age of hepatic cirrhosis. The grandmother, four brothers and sisters, and two children showed the same anomalies from youth; therefore eight persons in four generations. The cases resemble perfectly the first ones I described. They are to be reported more in detail by Dr. Haal, who has instituted far-reaching investigations concerning them.[1]

PATHOLOGY

We would digress too far were we to discuss more in detail the questions which arise concerning the subject under discussion. I shall, therefore, not enter upon the *pathologico-anatomical findings* in jaundice, but will only remark that the changes which develop in consequence of disturbances of the flow of bile in the liver are only in part to be referred to the *mechanical and chemical effects of the retained bile.* To a great extent they depend upon *secondary infection* of the biliary passages and upon the *decreased power of resistance* of the organ owing to the stagnation of bile, as opposed to the various deleterious agents capable of producing

[1] According to a communication of Dr. Haal, the number of erythrocytes was decreased, the excretion of uric acid increased, and the amount of iron in the urine decidedly increased.—Three additional cases of the same affection have just been described by v. Krannhals in the *Deutsches Archiv f. klin. Med.*, Bd. 81.

inflammation. To the most common signs of hepatic jaundice, as demonstrated in the enlargement of the liver and its icteroid discoloration, the dilatation of the biliary passages and the deposit of biliary stained masses in the hepatic cells and bile capillaries, with continued obstruction to the flow of bile, we may add the severe changes presented by the picture of *diffuse hepatitis* which, in its mode of development, presents certain peculiarities. These peculiarities are noted especially in the appearance of *focal necroses* in the parenchyma of the liver as well as in the marked prominence of *inflammation and processes of proliferation starting from the interlobulary biliary channels.*

As a rule, in the cases which perish under the picture of acute hepatic degeneration very conspicuous and peculiar changes of the organ are found. Upon opening the abdominal cavity in cases of well developed "acute yellow atrophy of the liver," the *small size and flaccidity* of the liver are conspicuous, and the organ, covered by coils of intestine, appears to have sunk deep down alongside the vertebral column. The weight of the organ may be less than one-half the normal; in consistence it is flaccid, withered, almost fluctuating, or doughy and friable. The serous covering looks wrinkled. The color of the liver is *yellow,* often uneven upon the cut surfaces, and between the yellow areas large or small *red foci* are found. These latter show a more advanced stage of the disease. After prolonged exposure to the air the cut surfaces are often covered with a wreath-like coating of *tyrosin crystals.*

Upon microscopic examination of such a liver the hepatic cells in the yellow areas are found to be in a condition of more or less advanced degenerative destruction: Cloudy, swollen, showing fatty degeneration, deformed, or transformed into detritus. In the red areas this detritus has been absorbed, and we note between the capillaries only a pale homogeneous, or striated, open-meshed connective tissue, in which, as the only remains of hepatic cells, isolated fat globules and granules of coloring material are found.

In the cases running a protracted course, in addition to the degeneration and destruction of liver cells, proliferative changes take place in the liver by which the destroyed hepatic tissue is replaced. Besides a proliferation of hepatic cells by indirect division of the nucleus, new formations appear, starting from the biliary passages and consisting of epithelial tubules, also processes of proliferation in the endothelia of the blood-vessels, and cell accumulation and connective tissue proliferation in the interstitial tissue. Such cases form transitional stages in acute and subacute forms of hepatic cirrhosis.

DIAGNOSIS

Before entering upon minute details of the treatment, we must consider the diagnosis and prognosis of jaundice.

The *diagnosis* of *simple jaundice* is readily made when the signs of the affection become marked. Its appearance after preceding digestive disturbances in previously healthy persons who, as a rule, present but slight constitutional symptoms, the absence of pain and of marked changes in the liver, above all its benign course, do not leave us long in doubt regarding the nature of the affection.

In my opinion, no sharp differentiation is possible of the severe forms of *icterus gravis* which run their course with acute degeneration of the hepatic parenchyma. Such differentiation has more of a prognostic than a diagnostic interest. Clinically, the limits must be drawn where the disturbances of hepatic function produce the symptoms of autointoxication. When the anatomical changes are considered, the demonstrable decrease in the size of the organ and the appearance of products of decomposition in the urine appear of greater significance.

The flooding of the organism with the products of decomposition of the liver, above all the appearance of *amido-acids* in the urine (and, since the discovery of Frerichs, particularly the excretion of *leucin* and *tyrosin*) must warrant the assumption of acute yellow atrophy of the liver. These substances are not found exclusively or even invariably in the above mentioned disease. By minute methods of investigation we have recently proven that the presence of amido-acids in the urine is not rare; they have been found in large quantities in the urine in gout, in pneumonia and in leukemia (Ignatowski).

In no other disease are such large quantities of amido-acids found as in acute degeneration of the liver. The urine here needs no preparation with sensitive reagents such as the recently employed β-naphthalin sulphochlorid. On the contrary, tyrosin is often directly deposited in the urine as a crystalline sediment in the form of characteristic bundles, sheaves, and strongly refractive needles. The more readily soluble leucin crystallizes in the urine only in the form of concentrically layered or radially striped globules.

The crystal forms may readily be confounded with urate salts. It is therefore advisable to ascertain the presence of these substances either by extracting them from the urine which has been boiled with alcohol, or by precipitating them with lead acetate and then removing the surplus quantity of lead with hydrogen sulphid, by which means we obtain a solution free from urate salts, in which after cooling leucin and tyrosin may crystallize.

The appearance of amido-acids, which have been recognized as the prior stages of urea, was formerly referred to a cessation of the formation of

urea in consequence of a deficiency of the hepatic function. According to my investigations, experimentally excluding the liver, as well as according to recent researches (Lang and others) concerning the prevention of amido-formation in the animal body, it appears likely that the splitting off of ammonia from the amido-acids may take place outside the liver, and only the synthetic change of ammonia into urea within the liver. As a consequence of disturbed hepatic function we may therefore assume at most an increased excretion of ammonia with a simultaneous decrease in the excretion of urea. This has actually been determined in some, but by no means in all, cases of acute yellow atrophy of the liver (Minkowski, Münzer, Fränkel, Senator, Soetbeer).

Diagnostically, however, this sign cannot be utilized. The degree of the excretion of urea and the total nitrogen excretion cannot readily be determined in cases of extreme insufficiency of the liver, because, in the first place, the ingestion of food as well as the digestion and absorption of food is decidedly limited; secondly, because a toxiferous proteid destruction which cannot be calculated is also operative; finally, because the excretion of nitrogen, owing to the severe damage to the kidney which usually occurs simultaneously, does not appear to be uniform. The relative increase of the excretion of ammonia may, however, be the result of an excessive production of acid in the organism, an *acidosis,* which is actually a frequent occurrence.

The abnormal acids which in such cases are usually found in the urine probably owe their origin to an increased destruction of tissue elements. We must include among these the *oxyacids* derived from the amido-acids by splitting off of ammonia, the excretion of which has been determined in the urine in acute yellow atrophy of the liver, also *hydroparacumaric acid* derived from tyrosin (the homologous *oxymandelic* acid found by Sehultze and Riess in the urine in acute yellow atrophy). Probably many other substances belonging to this category are also found.

Lactic acid (oxypropionic acid), which has been detected in the urine in some cases of severe degeneration of the liver, is probably derived from a corresponding amido-acid, the *alanin* which has also been found in the urine in these cases, as well as amidocaprionic acid and leucin. But the excretion of this (muscle) lactic acid is, perhaps, connected with a loss of liver function, for after experimental exclusion of the liver I noted remarkably large quantities of lactic acid in the urine.

Whether the *albumoses* and *peptones* which some authors have found in the urine in acute degeneration of the liver are also to be regarded as the results of the abnormal destruction of tissue elements is at least questionable. On the contrary, the increased excretion of *purin substances* (xanthin bases and uric acid) may undoubtedly be referred to an increased destruction of cellular nuclei. No special diagnostic significance can at present be attached to these varied occurrences.

The differentiation of simple forms of jaundice from jaundice due to gall-stones is of great practical importance. We cannot here enter into the details of the diagnosis of cholelithiasis. But that this condition is a cause of jaundice should be constantly borne in mind if icterus *develops with pain* or if *colicky attacks* of pain have previously been localized in the region of the gall-bladder, if the jaundice *develops very suddenly and soon disappears,* and if *repeated attacks of icterus* occur in the same person.

Above all, we must consider the possibility of incarceration of stone when the jaundice lasts for an *extremely long time.* Although, as has been stated, there are exceptions to this rule, simple jaundice very rarely lasts longer than six weeks. When the course is more protracted, *choletithiasis,* certain forms of *hepatic cirrhosis,* and, above all, *neoplasms* of the biliary passages come into question.

Marked entargement of the liver and spleen generally favors diffuse hepatitis.

The diagnosis of simple jaundice in an *elderly person* always demands the careful weighing of all the symptoms. In these cases neoplasms of the biliary passages and the head of the pancreas primarily come into question.

Close investigation of the various pathological conditions and their careful consideration will usually lead us to a correct diagnosis. But the diagnostic difficulties may sometimes be very great. Simple jaundice rarely fails to be recognized; much more frequently this is diagnosticated when instead another affection, especially cholelithiasis, is present.

PROGNOSIS

The prognosis in jaundice depends mainly upon its cause. In simple jaundice it is usually favorable; but, as already stated, every profound jaundice brings with it the danger of a more or less sudden hepatic auto-intoxication.

The greater the development of the typical clinical picture of acute yellow atrophy of the liver, the more serious the prognosis. The fact that this affection of the liver, up to the most recent times, was regarded by many authors as an absolutely fatal disease, and a termination in recovery as a signal proof of diagnostic error, was due to an erroneous idea that the process was of peculiar nature and produced parenchymatous degeneration of the organ which led to atrophy, and the connection of the disease with the milder forms of jaundice and of acute diffuse hepatitis was completely overlooked. If we consider that we are dealing only with different degrees in the intensity of the disease, it will not surprise us occasionally to find a case running a severe course yet terminating in recovery.

The prognosis in the individual case is decided mainly by the *severity of the general symptoms* and the *implication of the central nervous system.*

Even here the individuality of the patient must be taken into consideration. Leube reports a case in which the appearance of delirium and convulsions indicated icterus gravis; but the further course proved that it was merely the complication of simple jaundice with severe hysteria.

When a rapid decrease in the size of the liver is recognized with certainty, or when leucin and tyrosin appear in the urine, death may almost invariably be expected in a short time, usually in two or three days. But exceptionally a favorable outcome has been observed, even in these cases.

The appearance of the *hemorrhagic diathesis* is a most unfavorable sign. Occasionally the loss of blood is so great, particularly in hematemesis, as speedily to cause a lethal outcome.

The *renal function* is to be regarded as an important factor in prognosis. Even in mild cases, the intensity of the clinical symptoms and, above all, of the subjective symptoms depends chiefly upon the secretion of urine. So long as diuresis continues profuse, there is no immoderate accumulation of toxic products in the organism. In severe cases, the renal function decides the prognosis. Therefore, when the symptom of jaundice is added to a renal affection, it must be regarded as a very serious complication; the state of the kidneys in severe cases of jaundice is even more important, for the affection of the kidneys is here the consequence of a general pathologic condition, and, as a rule, the severity of the renal disease is parallel with the severity of the hepatic affection. The ominous symptoms attributable to the central nervous system which characterize the picture of hepatic autointoxication are, therefore, usually introduced by an excessive decline in diuresis and an extraordinary decrease in the excretion of nitrogen, and these may be regarded as an expression of the increased retention of toxic substances in the organism.

Finally, the duration of the jaundice is of great significance in the prognosis. Isolated cases have been observed in which jaundice lasted for several months and even for a year, running its course as simple benign icterus, the symptoms at last completely disappearing. As a rule, however, a prolonged attack of jaundice is accompanied by special dangers. We frequently attribute the jaundice to severe organic affections. Jaundice in itself will, in the course of time, lead to nutritive disturbances, emaciation and anemia, which may remain long after the icterus has passed away. If the flow of bile is not brought about in proper time, death usually occurs in the course of the first year, most often between the sixth and twelfth months. Cases of jaundice lasting several years are among the greatest of rarities.

TREATMENT

In the therapy of jaundice we must first take into consideration the fact that, in most cases, the greatest harm is wrought in the digestive organs, the port of entrance for the liver and biliary channels. When,

therefore, in a case of jaundice special circumstances indicate the direction of our therapy, it must be our first endeavor *to remove any deleterious agents still present in the digestive tract.* Various intestinal antiseptics that are recommended, such as salol, naphthol, etc., are in these cases much less effective than laxatives, of which calomel, rhubarb, and the saline laxatives are preferable for this purpose.

Since laxatives also stimulate peristalsis in the biliary channels, and thus assist in the evacuation of these tracts, they may also relieve the biliary channels of deleterious products which they may contain. Whether the biliary passages are disinfected by the introduction of substances which pass into the bile and there exert their power must still be regarded as doubtful. The salicylic preparations may possibly have an action of this kind. Certainly not only metals, such as lead and mercury, but also other substances introduced into the organism, for instance, methylene-blue and alcohol (Brauer), are excreted with the bile.

The next object of treatment is to promote the flow of bile into the intestine.

In fulfilment of this purpose, we attempt the following:

1. To decrease the consistence of the bile.

2. To increase the motive power which causes the propulsion of the bile.

3. To reestablish the normal hepatic function.

We first try to decrease the consistence of the bile by a profuse introduction of water. It is quite certain that the intake of water does not in the slightest degree influence the quantity of the hepatic secretion, as it does, for instance, the quantity of the renal secretion; water is by no means an integral constituent of the excretion of the liver, as it is of the kidneys. After water is taken into the stomach, the excretion of bile is increased, but this increase in the amount of bile does not exceed the increase of the biliary secretion which occurs after the ingestion of food. If, however, water is taken upon an empty stomach, as, for example, in mineral water treatment, the bile to a certain degree is diluted, for the increase of the bile is out of proportion with the simultaneous increase of the solid biliary constituents. There have been no accurate investigations of the effect of the introduction of water upon the viscosity of the bile. Strauss found the molecular concentration of bile, measured from its decrease at the freezing point, to be unchanged after the introduction of water.

It has been believed that the effect of water upon the consistence of the bile could be aided by the simultaneous administration of alkalies. But from the results of experimental researches carried out by Stadelmann and his pupils, alkalies do not appear directly to influence the composition of the bile. But it is possible that they may have a certain effect upon the mucin-like substance of the bile upon which its viscosity is chiefly dependent.

The effect of alkaline waters, such as those of Carlsbad, Neuenahr, Vichy, etc., has at present only an empiric foundation, and in cholangitis as well as in other affections of the digestive organs it is attributed to the general "anticatarrhal" action of these waters. But the influence of mineral water treatment upon intestinal peristalsis as well as upon the circulation of the blood and the liver is possibly even more important.

The propulsion of the bile may be promoted by any remedies which stimulate intestinal peristalsis. This probably depends upon the production of contractions in the musculature of the biliary passages, and especially of the gall-bladder, by which the bile previously secreted is more rapidly propelled onward. The action of remedies which have been designated as "cholagogues" is probably limited. As a rule, they are laxatives, such as calomel, rhubarb, aloes, jalap, podophyllin, evonynin and the like. It is possible that the action of olive oil and the oleate salts, of which eunatrol (sodium oleate) has lately been praised, is founded upon this. But it is difficult to understand why these substances in particular should be administered in jaundice when, without them, there is already a deficient absorption in the digestive organs of fats, fatty acids, and soaps. The apparent effect of olive oil in the treatment of gall-stones is probably due to the fact that the calcium and magnesium soaps which have been reduced to soft concrements by the oil have been mistaken for gall-stones.

The only cholagogues which actually increase the production of bile are the biliary acid salts. It is obvious that their administration in jaundice is irrational.

The outflow of bile from the liver may be further increased by remedies which strengthen the abdominal press, and thus compress the liver and the biliary passages. The action of the emetics formerly employed, such as apomorphin subcutaneously injected, probably depends upon this fact, but in general they are not advisable. These effects in a milder degree may also be brought about by physical exercises which favorably and simultaneously influence the portal circulation and the liver.

Careful massage of the abdomen may be very beneficial. Gentle massage of the hepatic region with the flat of the hand or by stroking in small circles, also vibration of the right hypochondrium, or the like, is generally preferable to that brought about by apparatus. Recently, however, the various forms of instrumental vibration and massage have been said to render good service. One author particularly advises shock of the entire body, because he himself was cured of a stubborn jaundice by a prolonged railroad journey.

The method, formerly advised by C. Gerhardt, by which the extended gall-bladder was encircled with the fingers and expressed has quite properly been abandoned, since it is of doubtful utility and by no means devoid of danger.

On the other hand, faradization of the abdominal wall, which occasion-

ally is very beneficial in the treatment of jaundice, is still less harmless. It appears to make no difference whether, as Gerhardt originally advised, one electrode is placed in the region of the gall-bladder and the other horizontally opposite and to the right of the vertebral column, or whether the current is extended to other areas of the abdominal wall.

Active gymnastic exercises are said to promote the flow of bile, particularly deep methodical respiration by which a kind of auto-massage of the liver is attained.

Injections of large quantities of water into the colon are very valuable for the purpose under consideration, since they produce an effect by stimulating peristalsis as well as by direct pressure upon the liver and the gall-bladder. According to Krull, injections of cold water (12° to 18° R.) more powerfully stimulate the intestinal mucous membrane. It is, however, frequently desirable to avoid irritation of the intestine; in such cases water of the same temperature as the body is slowly injected into the bowel with the least possible force. By this means the intestine is thoroughly cleansed and there is simultaneously an increased absorption of water which, for various reasons, is very desirable in jaundice.

In order to restore the normal hepatic function, the best method is to assist the curative processes in the diseased organ by decreasing the labor thrown upon the organ as well as by facilitating the circulation of the blood therein.

The labor required of the liver depends mainly upon the quantity and composition of the food. The necessity of sparing the diseased organ therefore primarily necessitates a limitation of the entire food mass. The fulfilment of this purpose, particularly at the beginning of the disease, is in most cases greatly facilitated by existing anorexia and repugnance to food. Often it is much more difficult to prevent the limitation of food from exceeding the boundaries necessary to maintain the body-weight and the activity of the organism.

As to the composition of the food, it must be remembered that the liver is implicated in the digestion of all organic food products, carbohydrates and fat as well as albuminates. As experience teaches us, however, the absence of bile in the intestine results chiefly in a deficient absorption of fats. It must be borne in mind also that the toxic products which cause hepatic autointoxication most likely originate in changes which proteid substances undergo. In jaundice there is almost always an instinctive repugnance to fat and meat. Therefore, in regulating the diet in jaundice, fats must be limited as far as possible, and when practicable these should be given only in a well emulsified form, as in milk; the ingestion of albumin should be kept within moderate limits and not exclusively in the form of meat; and the total amount of food, principally carbohydrates, should be given in an easily digestible form.

A plentiful supply of water in jaundice is desirable for more than one

reason; above all we must here consider that the acceleration of the circulation of the blood in the liver and the absorption of water are simultaneously combined with the dilution of all those substances which, in their passage through the liver, are capable of damaging the cells of this organ.

Alcoholic drinks must be excluded, as there can be no doubt of the deleterious effect of alcohol upon the liver. Other irritants such as pungent spices (pepper, mustard, red pepper, and the like) must also be avoided, since experience has demonstrated their unfavorable effect upon the liver, and also because the relations of the portal vein circulation make it appear probable that, after the gastric mucous membrane, the hepatic cells are most readily damaged by the action of these substances.

Moreover, it has lately been shown experimentally by Kobert and his pupils (Carlau, Jürss) that substances contained in mustard, pepper, nutmeg and parsley (oil of mustard, piperin, myristicin, apiol) exert an especially toxic effect upon the liver which somewhat resembles the effect of phosphorus.

In the nutrition of patients with jaundice the following dietary is advisable: Milk, milk soups, broths, fruit soups and flour soups, very little meat (150 to 200 grams), without fatty sauce and with but little spice; wheat bread, stewed fruit, and light starchy food; tender vegetables in moderate quantity and prepared without fat, especially as purée; weak tea, abundance of ordinary water, or alkaline and carbonated mineral waters, and, if desired, fruit syrups. Naturally, in choosing the diet, we must, as far as possible, consider the individual circumstances.

The rapidity of the circulation of the blood through the liver is powerfully influenced by the ingestion of food, for the amount of blood which flows through the liver depends chiefly upon the quantity of blood which passes through the roots of the portal vein. Reflex hyperemia of the digestive organs following the administration of food always results in an increased supply of blood to the liver; but while the resorption of digestive products caused by the administration of solid food leads to an increased exercise of the functions of the hepatic cells, water causes only an advantageous increase in the rapidity of the circulation and in the nutrition of the liver, without markedly increasing the labor of the organ. Herein probably lies the explanation of the favorable results in jaundice from the administration of water, especially of the mineral water cures, and for this reason it appears perfectly reasonable that the use of mineral water should be restricted to the early morning hours when the stomach is empty.

It is obvious that the various physical curative methods previously described, as well as the stimulation of intestinal peristalsis by laxatives, will quicken the circulation of the blood in the liver. And here the chief cause of their effect in jaundice is more readily recognizable than in the mechanical stimulation to the flow of bile.

Finally, one of the principal aims of the treatment must be described, namely, the protection of the organism from the injurious consequences of the impediment to the flow of bile. In pursuance of this aim, the following points are, above all others, necessary:

1. To increase the excretion of the accumulated biliary constituents by stimulating the vicarious activity of other glands.

2. To combat the digestive disturbances due to the absence of bile from the intestine.

3. To treat symptomatically the disturbances arising from the presence of biliary constituents in the blood.

To promote the excretion of the biliary constituents circulating in the organism, the activity of the kidneys comes chiefly into consideration. Here also the plentiful intake of water is the remedy by which these harmful substances may most readily be washed from the body. The diuretic action of the salts which are contained in mineral waters is also beneficial for this purpose. In special cases, particularly with threatening autointoxication, more active diuretics, such as caffein, diuretin, etc., are sometimes of use.

In most severe cases of cholemic or hepatic autointoxication, when the condition of the patient will not permit the copious introduction of water either by the mouth or by the bowel, we must attempt by subcutaneous or intravenous infusions of normal salt solution to restore the diuresis which has threatened to cease.

The excretion of biliary constituents by other secretions, for therapeutic purposes, scarcely comes into question. Biliary constituents are only excreted in the urine and in the sweat. It has been supposed that the yellow discoloration of the body linen frequently observed in cases of jaundice was due merely to the absorption of coloring material from the desquamated yellow-stained epithelia of the skin. But Fr. Müller and Leube have obtained the undoubted reaction for bile in sweat produced by injections of pilocarpin. However, the amount of biliary constituents excreted in this manner is very slight. The favorable influence of warm baths is due rather to their effect upon the circulation of the blood and the entire metabolism, and especially upon the excretion of urine, than upon the stimulation of excretion by the skin.

Biliary constituents pass into the other glandular secretions only when there are pathological changes in the secretions or an admixture of inflammatory exudates therein. In serous effusions there may be a very decided admixture of biliary constituents. In cases of jaundice in which larger effusions of this kind are present, it is more often advantageous to relieve the organism of biliary constituents by aspiration and the evacuation of these effusions than to endeavor to bring about their absorption by drugs.

Digestive disturbances due to the lack of bile in the intestine are most actively combated by such regulation of the diet as has been described.

Good service may also be rendered by laxatives, by mineral water treatment, by intestinal irrigation, and, in suitable cases, by gastric lavage. For the disagreeable taste complained of in the latter, and the subjective symptoms on the part of the stomach, hydrochloric acid or carbolic acid may be employed.

In regard to the symptoms, the itching of the skin produced by the flooding of the organism with biliary constituents is one which requires special treatment. Among remedies which have proven effective and which should be enumerated here are the following: Cold ablutions, perhaps with the addition of vinegar, citric acid, soda, aluminum acetate or a one per cent. solution of carbolic acid; inunctions with a 4 to 5 per cent. carbolic acid salve, 10 to 20 per cent. bromocoll salve, or with a 2 to 3 per cent. alcoholic menthol solution.

In stubborn cases the internal administration of potassium bromid (2 grams twice daily) and injections of pilocarpin (0.01–0.02) or of atropin (0.0005–0.001) are recommended. Often we require narcotics (morphin, chloral hydrate, and the like). The frequent use of warm baths is very beneficial.

The symptoms of severe cholemic intoxication indicate such methods of symptomatic treatment as are based on the general laws of the treatment of poisoning.

CHRONIC INFLAMMATION OF THE LIVER

By E. STADELMANN, Berlin.

Under the name of chronic inflammation of the liver we include all those inflammatory processes in the liver which are distinct from acute inflammation, hepatic abscess. Usually the term "chronic interstitial hepatitis" is also applied to them, and this designation simultaneously indicates that the condition consists chiefly in an inflammation of the connective tissue. Under such a conception, however, difficulties arise. Undoubtedly there is an increase of the connective tissue in every chronic inflammation of the liver, but this is by no means to be regarded as the primary element. On the contrary, eminent authorities—I shall here mention only Weigert and Ackermann—have affirmed that a degeneration of the hepatic cells is the primary, and hypertrophy of the connective tissue only the secondary, element. Although this view unquestionably has much in its favor, and receives abundant support from the fact that a number of poisons which produce chronic inflammation in the human organism—I shall only mention alcohol, phosphorus and arsenic—positively, directly, and primarily attack the liver cells, producing therein contraction, degeneration, fatty infiltration, fatty degeneration, granular cloudiness, and also cause the disappearance of nuclei and coagulation necrosis, etc., nevertheless, in my opinion, schematism would carry us too far if we were to assume that all increase of the connective tissue, which is the characteristic of chronic hepatic inflammation, is only secondary, that is to say, passive, and not also active. I do not believe that this connective tissue increase is produced solely by the primary destruction of the hepatic cells in such a way that by distention, distribution, and proliferation it fills the spaces present. On the contrary, I think we must take the standpoint that many of the etiological factors of chronic interstitial hepatitis act upon the hepatic cells as well as upon the interstitial connective tissue, at one time chiefly upon one, at another time chiefly upon the other, and, in the main, with such effect that the hepatic cells contract under this influence and perish, while the connective tissue, on the contrary, increases, that is, hypertrophies. I desire, therefore, to express my conviction that in many cases of chronic interstitial hepatitis the connective tissue hypertrophy is not merely a passive one, but is also active, and that it is incorrect to exclude by the name "chronic interstitial hepatitis" our conception

of primary degenerative parenchymatous conditions. Important proofs of the correctness of this view are at hand. Degeneration of the hepatic cells, as well as connective tissue hypertrophy, is undoubtedly present in advanced hepatic cirrhosis—this is proven by the investigations of Ackermann (1), Hartung (2), and others—but in itself it is not sufficient proof; for why should not the cell degeneration in these advanced stages of disease be a secondary effect produced by the pressure of the hypertrophied connective tissue? The reason must be found in the stage of onset of the process. Is the affection of the cells, or the connective tissue proliferation, primary? In three such cases which he investigated Brieger (3) found both conditions. Orth states in his text-book that new connective tissue formation is often found without a trace of cellular change. A number of other authors express the same opinion. Minute microscopic investigation in chronic interstitial hepatitis shows that connective tissue proliferation varies in different cases. While in one instance the proliferation, in the main, takes place in the interlobular connective tissue encircling one or more hepatic lobules (annular, insular form, that is, monolobular and multilobular cirrhosis), at other times it is more or less distributed within the individual lobes and between the individual hepatic cells (the intralobular, that is, the intracellular form). Connective tissue is also sometimes deficient in cells, fibrous (older foci), sometimes rich in cells (recent foci). The liver cells themselves sometimes show decided degenerative processes, such as infiltration with fat and bile, or the cells are small and contracted; in other cases they are nearly normal, or even enlarged. In almost all processes of the liver, and going hand in hand with new connective tissue formation, net-like canals are found with lumen which are in part to be considered the remains of normal biliary passages, partly as newly formed bile ducts, or their development may be attributed to a transformation of the epithelia of the true hepatic cell trabeculæ which are flattened, so that, according to this last conception, they are not related to the actual biliary passages, nor do they possess special walls (Aufrecht). It is only certain that these canals have a lumen, and that they may be injected by way of the biliary system (Ackermann). The macroscopical appearance of the liver varies in different cases; sometimes the organ is enlarged, sometimes decreased in size; occasionally smooth, at other times nodular; sometimes it is of normal consistence and feels coarse and hard, at other times it is more doughy (fatty degeneration); sometimes the liver is greatly stained by bile, at other times not; sometimes the organ is dark-brown in color and greatly pigmented, at other times whitish and massively infiltrated with fat.

We shall now briefly discuss the different forms of hepatitis, and their mode of origin.

A. THE VARIOUS FORMS OF CHRONIC HEPATIC INFLAMMATION

The various findings mentioned above have led to the differentiation of many individual forms of hepatic cirrhosis, and the greatest schematic development has been attained in France where some authors have elaborated eight, ten, and even fifteen different forms of hepatic cirrhosis. This number will be still further augmented if we take into consideration the etiology of new connective tissue formation and the clinical course of the disease.

Under these circumstances we may maintain comprehensively that new connective tissue develops in the liver from the following causes:

1. Local, chronic foci of inflammation develop around *foreign bodies* (particles of coal dust) which have entered the liver; in the surroundings of *tumors*; around *parasites* (echinococcus); from implication of the liver, from *chronic inflammatory processes in neighboring organs* (stomach, pancreas); from trauma which produces local injury to the liver, and when, in consequence of a primary hepatitis, a secondary connective tissue proliferation of greater or less extent also takes place in the hepatic tissue. To this category also belong the small interstitial proliferations caused by *foci of bacteria* from various *infectious* diseases (tuberculosis, variola, scarlatina, morbilli, diphtheria, etc.).

2. In connection with the abundant and regular use of pungent *spices* (particularly curry) and the abuse of drastics (Cantani) after *gout*. These modes of development must still be regarded as unproven. The same is probably true of

3. The appearance of cirrhosis after *portal vein thrombosis*, to which Botkin and Solowieff (4) have called attention. Solowieff has endeavored to prove Botkin's view experimentally, but these experiments are by no means conclusive. Thrombosis of the portal vein without hepatic cirrhosis is unquestionably observed very frequently in man, and where these occur simultaneously the assumption is obvious that cirrhosis was primary, and thrombosis of the portal vein secondary. Simple occlusion of the portal vein leads to atrophy of the liver.

4. Cirrhosis in connection with *arteriosclerosis* is maintained by Duplaix (5) who, in fact, does not regard hepatic cirrhosis as an independent local disease, but as an affection which accompanies systemic disease. There is so little upon which to base this view that discussion hardly appears necessary. Arteriosclerotic changes may occasionally be found in the hepatic vessels, and may lead to local interstitial proliferation, but true hepatic cirrhosis in consequence of arteriosclerosis may be regarded as unproven.

5. In chronic states of *stasis hyperemia of the liver* in consequence of continuous pressure from the dilated blood-vessels, an atrophy of the

hepatic cells and hypertrophy of the connective tissue, especially about the central portions of the lobules (hepatic vein), which, however, may also penetrate into the atrophied hepatic cell trabeculæ and even include the interlobular spaces (nutmeg liver), so that at last the picture of atrophic hepatic cirrhosis with ascites, etc., develops. To this category probably belong the chronic perihepatitis and pericardial pseudo-cirrhosis lately described by several authors (H. Rumpf (6) and Fr. Pick (7)). We shall later revert to these forms of disease, and describe them somewhat more in detail.

6. The quite frequent coincidence of hepatic cirrhosis and *tuberculosis* is remarkable, particularly tubercular peritonitis. Wagner (8), Moroux (9) and others report numerous instances of such simultaneous occurrence. Wagner's explanation appears to me to be correct, namely, that cirrhosis is the primary condition, and tuberculosis the secondary; nevertheless, there are distinguished authors (Weigert) who hold a different opinion, and who maintain that the inverse relation of these diseases to one another is the true one. According to these, therefore, tuberculosis is the primary condition, and causes the cirrhosis.

Tuberculosis of the liver, naturally, is frequently observed without a simultaneous cirrhosis. The tubercular nodules in the liver frequently lead to no inflammatory proliferation of the surrounding tissue worth mentioning. In other cases, however, this is very striking, and when a great number of tubercular nodules, which naturally must have existed for a long time, are present, the picture of diffuse interstitial proliferation develops, and presents some similarity to that of hepatic cirrhosis. Contraction of the organ in this case is very rare, probably because the underlying condition (tuberculosis) early causes death, for tuberculosis of the liver is but a phase of general tuberculosis. We do not deny the existence of such forms, but certainly primary cirrhosis with the subsequent development of tubercles is more frequent. Occasionally we see this combined with more or less decided fatty infiltration of the liver cells. That tuberculosis may lead to fatty liver has long been known, and cannot be regarded as singular. At one time this, at another time that, of the pathologic conditions enumerated preponderates, and thus varying pictures are presented. We are, however, scarcely justified—and this is evident from the above brief discussion—as the French maintain (Hanot and Gilbert (10) and others), in differentiating this form of cirrhosis as a special one, and then proposing numerous subdivisions—not less than six. This is certainly carrying schematism too far.

7. Here we must mention the *malarial liver* which in Germany is quite rare. We therefore quote the following description from French and Italian literature, without permitting ourselves the right of criticism as to the correctness of these findings. It is well known that in profound malarial infection a large amount of pigment is formed from the destroyed cor-

puscles, and this is deposited in various organs, particularly in the spleen and liver. The pigment enclosed by leukocytes is sometimes found in the hepatic capillaries, producing thrombosis therein, sometimes within the liver cells, and to some extent also in the interstitial tissue. The liver assumes a rusty brown or dirty grayish-brown appearance. The pigment sometimes gives a reaction for iron (melanin), sometimes not. The liver is rich in bile—as in mild jaundice—enlarged, hyperemic, smooth, and painful upon palpation.

In the acute cases proliferation of the interstitial tissue does not occur, as is the case in chronic malaria. In the latter disease marked enlargement of the liver is found, partly in consequence of hyperemia, partly due to an increase of the parenchyma. The liver cells are large, swollen, opaque, with a large nucleus frequently beginning to segment. If the course of the disease is prolonged, from this stage a second develops in which the liver becomes atrophic. The liver cells degenerate, the connective tissue is but little increased, the organ is flaccid and smooth. Sometimes, however, a different condition is found (Kelsch (11) and Kiener) and the liver is more voluminous, is soft, irregular, covered with small nodules, which are whitish, or golden yellow to greenish, of the size of a pea, projecting from the reddish parenchyma. In these nodules the cellular trabeculæ are greatly thickened (to four times their normal size). The individual cells are very large and opaque, and show one or more large nuclei. The normal arrangement of the hepatic lobules is broken up, the peripheral hepatic trabeculæ being centrally flattened by the pressure of the hypertrophied tissue. Sometimes these nodules resemble adenomata. In about one-third of the cases (probably the oldest forms of the disease) we then observe more prominent interstitial proliferations starting from the interlobular spaces, and resembling the process in ordinary hepatic cirrhosis.

These forms and stages of hepatic disease may be combined in one and the same case to a varying extent and thus produce very different pictures. According to Ughetti (12) the malarial liver in the south of Italy differs nowise from ordinary hepatic cirrhosis, and does not present the peculiarities described by Kelsch and Kiener.

8. Similarly as in malaria, hepatic cirrhosis also occurs in *diabetes mellitus*. Some authors have included both forms—this and the preceding—under the term "cirrhoses pigmentaires." Cirrhosis in diabetes—at least in Germany—is a rare disease; this was first described by Hanot and Chauffard (13) as cirrhose pigmentaire hypertrophique. The patient shows marked cutaneous pigmentation—similar to that in Addison's disease—with cachexia and moderate diabetes. The liver is greatly enlarged and indurated, and shows decided pigmentation as well as profuse cirrhotic connective tissue proliferation. The pigment gives a reaction for iron, and shows itself to be closely related to hemoglobin. Hence we may

conclude that in the organism of the patients affected great and long-continued destruction of red blood-corpuscles plays an important rôle. Quincke (14) called attention to the fact that in diabetics there is "siderosis," i. e., a deposit of iron-containing pigment in the different organs. Occasionally this occurs in consequence of hepatic cirrhosis. Upon the basis of Quincke's investigations, however, the view so widely current in French literature, that siderosis of the liver is the cause of cirrhosis, must be rejected. We fail to see why a diabetic should not have hepatic cirrhosis, and, on the other hand, why a patient with hepatic cirrhosis should not contract diabetes. I am unwilling to acknowledge any causal connection between these diseases. In fact, patients with hepatic cirrhosis and a simultaneous diabetes by no means always show the above mentioned pigmentation. Why diabetes leads to hemosiderosis is not quite clear. Perhaps acidosis, the production of oxybutyric acid, leads to the destruction of the red blood-corpuscles in the organism. I believe this a subject well worthy of discussion. That autointoxication occurs in the course of severe diabetes, in consequence of acidosis, that is, the production of oxybutyric acid (coma diabeticum), is, in my opinion, a positive fact. That the circulation of abnormal products of metabolism in the blood must exert a deleterious influence upon the red blood-corpuscles appears to me to be quite obvious. But we would digress too far to follow this thought at the present time. I desire only to mention that in two patients with diabetes and distinct hepatic cirrhosis, whom I have under observation at the present time, the one without acidosis presents no sign of cutaneous pigmentation, while the second, who has been constantly observed for about three years, and who, in spite of a very strict diet, has about 4 per cent. of sugar in the urine, and is passing three liters in twenty-four hours, has severe acidosis and presents the typical picture of diabète bronzé. The former of the patients just mentioned succumbed after a continuous observation of several months, and in him ordinary cirrhosis hepatis was found without any peculiarities. The amount of sugar in the urine, which at first varied between 7 and 8 per cent. (the amount of urine could not be definitely determined, as the patient was careless), then gradually decreased, notwithstanding the fact that no dietetic measures could be carried out with the patient, and during the last two or three months of his life sugar had entirely disappeared. Besides cirrhosis and diabetes there was also decided cystitis, but no nephritis; periodically considerable blood was found in the urine, and the autopsy proved that this originated in the bladder. The urine never showed the iron chlorid reaction. At the autopsy the pancreas was found to be perfectly normal.

The second patient exhibited marked cutaneous discoloration (grayish-brown) in different areas (face, hands, testicles, penis, skin of the abdomen). The pigmentation has increased but little in the last few years. The liver is large, reaching to the umbilicus, is tough, smooth, and reveals

a sharp margin; there is no tendency to contraction. The spleen is not enlarged. For about a year and a half the patient has employed alkalies (sodium citrate) continuously, maintains his weight at about 60 kilos, and follows his occupation. There is no jaundice, no ascites, no edema. That the disease, as reported, rapidly leads to cachexia and causes death from ascites and edema, therefore does not appear to be true in all cases. Certainly there has been no apparent change in the condition of my patient for about three years, and the liver invariably shows the same abnormalities.

In diabetes not only the hypertrophic, but also the atrophic, form (Hanot (15) and Schachmann) is occasionally observed.

That there is an undoubted and distinct pigmentation of the skin in these cases, among which the case of my patient above mentioned belongs, and not, as Quincke believes, chiefly a cachexia, I positively maintain from my own experience.

Siderosis with hepatic cirrhosis also occurs in other diseases without diabetes; for instance, in pernicious anemia (Quincke) and phthisis pulmonalis (Marchand).

9. The Italians, for instance Galvagni and Ughetti (16), have described a form of hepatic cirrhosis as "*hepatitis interstitialis flaccida.*" But our experience regarding these observations is at this time so slight that we can do no more than mention the name.

10. The French (Hutinel, Sabourin (17)) have several times described a type of hepatic cirrhosis as "*cirrhose graisseuse,*" and have differentiated it as a distinct form. This is said to be characterized by a marked fatty infiltration of the hepatic cells as well as the distinct proliferation of the interstitial tissue. The liver is sometimes increased in size, at other times small; this form is found in alcoholics as well as in the tubercular. I do not recognize it as a special variety. Any one who frequently examines cases of hepatic cirrhosis knows that the amount of fat in the cells varies within wide limits, and occasionally may be enormous. Here we have probably only the combination of cirrhosis with fatty liver. Which affection is the primary one is of less importance; that alcoholism produces fatty degeneration as well as cirrhosis has long been known, and that fatty liver as well as interstitial proliferation may be observed in tuberculosis has just been stated under paragraph 6.

11. Chronic inflammation of the liver in consequence of the chronic effect of poisons. When *alcohol* is habitually taken by man in large quantities, it not only produces fatty infiltration of the liver but also hepatic cirrhosis. This statement may be regarded as a positive fact. The only question is whether chronic alcoholism produces different forms of interstitial hepatitis or only that form which we designate as Laennec's atrophic hepatic cirrhosis, or also granular atrophy. Later we shall minutely discuss the fact that, chiefly for clinical, and less for anatomical, reasons,

we differentiate two forms of hepatic cirrhosis, namely, (a) the above described atrophic or Laennec's cirrhosis, and (b) hypertrophic hepatic cirrhosis. Some authors, particularly Rosenstein (18), assume that the latter are not attributable to the abuse of alcohol, while most authors agree in regarding alcohol as the *causa peccans*. The question is so important that it must be discussed somewhat more in detail.

Hepatic cirrhosis is most often caused by spirits, and it is especially noteworthy that the quite large number of children mentioned in literature as addicted to alcohol, and who are found with hypertrophic hepatic cirrhosis, are made drunkards chiefly by the deplorable ignorance of their parents and relatives. But beer and wine also cause hepatic cirrhosis, although much more rarely.

In France the immoderate use of absinthe is often alleged to be the cause of hepatic cirrhosis. The following is shown by statistics: That in about one-third of all cases, according to Frerichs (12 patients in 36) as well as Bamberger, the immoderate use of alcohol may be regarded positively as the cause. Price (19) found among 142 cases of hepatic cirrhosis 108 occurring in men and 34 in women (i. e., about 3 to 1). Abuse of alcohol was positive in 72 cases, probable in 8 or 9, unmentioned in 50, and positively denied in a few. But in these statistical compilations the two forms of the disease have not been separated. Mangelsdorf (20) found in 49 cases of hypertrophic cirrhosis that 19 might be etiologically referred to alcoholism, in 5 alcohol was suspected, and in 17 it could not be excluded. In only 5 cases was there no history of alcohol or syphilis, the latter affection being the positive cause in 3 cases, and likely in 2. With the widely prevalent use of alcohol by all classes of the population, it is extremely difficult to prove, in the individual case, that it has had a deleterious effect and has caused the disease. What is regarded as much by one person another considers very little. One person who takes habitually a certain quantity of alcohol regards himself as strictly temperate, another who drinks the same quantity suffers from pangs of conscience, and regards himself as an alcoholic. But the same quantity of alcohol has a baneful but varying effect upon different persons. Here we cannot reckon toxicologically by the kilo of weight. One person on a moderate daily amount of alcohol is attacked by hepatic cirrhosis; another worships Gambrinus, Bacchus, and the god of alcoholic spirits, and goes unpunished all his days. But the cases which Mangelsdorf describes, and others which I have found in literature, are so positive and conclusive that there can be no question as to the influence of alcohol in the development of hypertrophic hepatic cirrhosis. The consumption of from 3 to 6 liters of wine daily is, nevertheless, a large amount, particularly if we refer to its use by women. These reports are so conclusive that we can hardly reconcile with them the view of Rosenstein (20 a and 65) that, in his cases, alcohol as an etiologic factor could be excluded. We must emphasize that Rosen-

stein's view is of great importance, since he has seen a large number of cases of hypertrophic hepatic cirrhosis, and it cannot be denied that the frequency of the occurrence of these forms varies greatly in different localities. In spite of the immoderate use of spirits and the great prevalence of atrophic hepatic cirrhosis in Berlin, typical forms of hypertrophic hepatic cirrhosis are quite rare. Therefore, we conclude that some unknown factor besides alcohol must be operative to produce hypertrophic hepatic cirrhosis.

How alcohol causes hepatic cirrhosis has not yet been made clear. It is possible that it first produces inflammation in the vessels while it is passing in a concentrated form from the stomach and intestines into the portal vein vessels, and perhaps it has also a directly deleterious influence upon the hepatic cells. We cannot say more than "perhaps." None of the views previously expressed are positive, and each requires proof. It is said that alcohol is not excreted by the hepatic cells, for, according to the researches of Weintraud, even after the administration of large quantities it cannot be detected in the bile.

The results of Weintraud's investigations are diametrically opposed by the recent experimental inquiries of Brauer (*Zeitschr. f. physiolog. Chemie,* XL, H. 3 und 4). This author, after administering large doses of alcohol to dogs, readily demonstrated the presence of alcohol in the bile, besides quite decided amounts of albumin. These findings should be of great significance in proving the correctness of the view above expressed that alcohol enters the liver unchanged, passes into the bile, and directly irritates and damages the hepatic cells. This continuous damage would finally lead to the destruction of the cells and to the development of hepatic cirrhosis. Alcohol is also found in the urine, but less abundantly than in the liver. Besides being affected by cirrhosis, many alcoholics also suffer from nephritis, and these affections may quite properly be referred to the same cause, namely, alcohol. The secretion of the milk glands, however, does not contain alcohol (Klingemann), consequently nurslings, at least, are protected from the consequences of the abuse of alcohol on the part of their mothers and wet-nurses.

Alison's (21) statistics are also interesting; he found in the country among farm hands the proportion of cirrhosis to alcoholism as 1 to 85; in the city, however, with a sedentary mode of life, it was 1 to 25, and with great bodily activity the number of city cases also decreases greatly, i. e., to about 1 to 42. Therefore, we should send our alcoholics to the country and assign them to strenuous corporeal labor if we desire to protect them from hepatic cirrhosis, and this would not even be injurious; but it would prove as difficult to enforce this prophylaxis as to induce them to renounce their alcohol.

In comparison with alcohol as the cause of hepatic cirrhosis other poisons are insignificant.

In man *phosphorus,* more than any other, destroys the hepatic cells

and produces marked fatty infiltration. It frequently causes the anatomical picture of acute yellow atrophy, but rarely true hepatic cirrhosis.

Arsenic, antimony and *chloroform* are also said to produce hepatic cirrhosis in man, and, according to Langowoi (22), this is also true of cantharides. Here it seems fitting to consider somewhat minutely the *experimental investigations* which have been made in the hope of solving the many questions under discussion which have just been enumerated. An attempt was made to produce hepatic cirrhosis in animals, and for this purpose various poisons, especially alcohol, phosphorus and chloroform, were used. Unfortunately, as we must admit at the outset, most of these endeavors have been fruitless.

It has been impossible to produce hepatic cirrhosis in animals by administering alcohol. This is shown by the invariable negative trials of Strassmann (23), Afanassiew (24) and v. Kahlden (25). Fatty liver was induced, but not hepatic cirrhosis. In my opinion these results contradict the earlier and positive reports of Dujardin-Beaumetz, Pupier, Strauss and Blocq. Afanassiew claims that he has produced degeneration of the hepatic cells by prolonged and large doses of alcohol, especially in rabbits, while v. Kahlden denies such a pathologic finding.

Perhaps the toxic effect of alcohol with large, individual doses was not long enough continued.

H. Mertens maintains that he has lately produced typical hepatic cirrhosis in rabbits by keeping the animals continuously in an atmosphere permeated with alcohol.

According to Mertens (26), chloroform in subcutaneous injections has a much more powerful effect. He was able to produce typical hepatic cirrhosis, and to determine that the liver constantly became harder, more granular, and more contracted. The liver cells suffered first; they became opaque, atrophic, and showed fatty degeneration, being permeated with vacuoles. Later, starting from the portal vessels, connective tissue developed which became diffused through the hepatic lobules toward the center (the hepatic veins). By the kindness of Professor Heymanns in Ghent, where Mertens worked, I was able to obtain sections of such livers for examination. These livers showed typical granular atrophy with the above described microscopic findings. Nevertheless, I am very skeptical in regard to experimental investigations in rabbits on account of my own extensive experience. These animals frequently have diseased livers, and consequently we are easily led astray in regard to, at least, the early stages of cirrhosis.

I do not altogether countenance the investigations of Boix (27) who, by the introduction of small amounts of butyric acid (for three months), acetic acid (for thirty-five days), and also of lactic acid and valerianic acid, produced typical atrophic hepatic cirrhosis in rabbits.

Recent experiments have cast a doubt upon the apparently positive production of phosphoric hepatitis observed by Wegner. Krönig (28), as

well as Ziegler and Obolensky (29), found almost invariably marked epithelial degeneration, but the inflammatory phenomena were only moderate, and, according to my investigations, the conditions resembled those produced by toluylendiamin. Ziegler and Obolensky expressly state in their report: "It appears impossible, from the investigations at hand, to draw conclusions from poisoning by phosphorus and arsenic as to the genesis of hepatic cirrhosis in man. At present the latter can only be determined by the histologic investigation of cirrhotic livers."

Aufrecht (30), it is true, holds a different opinion. He maintains that he has seen connective tissue hypertrophy follow phosphorus poisoning without any epithelial degeneration. I reiterate the fact that Aufrecht experimented on rabbits, but, as mentioned above, in these animals such experiments are not conclusive.

We have still to mention four other forms of chronic inflammation of the liver, which will be explicitly described later:

12. Cirrhosis hepatis, that is, Laennec's cirrhosis, which has several times been mentioned.
13. Hypertrophic hepatic cirrhosis.
14. Biliary hepatic cirrhosis.
15. Syphilitic hepatic cirrhosis.

From a clinical rather than from a pathologico-anatomical standpoint it appears necessary to differentiate the two fundamental forms of hepatic cirrhosis, (a) the atrophic and (b) the hypertrophic hepatic cirrhosis, as two contrasting types, between which are numerous transitional forms; therefore they will be separately considered.

B. CIRRHOSIS HEPATIS—LAENNEC'S CIRRHOSIS—GRANULAR ATROPHY OF THE LIVER

This form of hepatic cirrhosis receives its name, which was first given it by Laennec, from the fact that in advanced stages it leads to contraction which causes the anatomical condition to resemble the granular atrophy of the kidneys. Etiologically this form, as described above, may chiefly be attributed to the abuse of alcohol, hence the affection preponderates among men. Although all clinicians are unanimous on this point, it must, nevertheless, be stated that there are cases of hepatic cirrhosis in which the abuse of alcohol cannot be proven, nor can any of the causes enumerated for the appearance of the affection be found. The question, therefore, arises whether or not, as with alcohol, other toxic products in large quantities (products of metabolism) may not occasionally find their way from the intestine into the portal vein system, and there exert a deleterious influence upon the liver which may result in hepatic cirrhosis. We all know that in the human organism an antitoxic function is quite properly ascribed to the liver, and the inference is obvious that occasionally under

an increased virulence of the toxins which are normally brought to the organ, and considering the amount of labor to be performed, this antitoxic power may not be exerted. A possible support for this view may be found in the latest investigations of Brauer (*Zeitschr. f. physiol. Chemie,* Bd. 40, Heft 3 u. 4) who, after administering relatively large quantities of methylene blue to animals by the mouth, soon after found it in the bile, sometimes even in larger quantities than in the urine. Methylene blue, therefore, in concentrated form, passes from the digestive tract into the liver, from which it is excreted into the intestine by means of the bile; therefore it passes through an intermediary circulation. Similar processes might be considered in the case of toxins, and the investigations of Brauer at least prove that this is not impossible, but actually occurs. Perhaps the unproven reports of the production of hepatic cirrhosis after the use of pungent spices (particularly curry), after the frequent administration of drastics (Cantani), after taking the extract of male fern (Grawitz), after gout, etc., may thus be satisfactorily explained; nevertheless, we are forced to conclude that the individual predisposition here also plays a significant rôle.

PATHOLOGICAL ANATOMY

The most important points have already been enumerated. In the advanced stage the organ is contracted, small, and very hard, so that upon section with a knife a grating sound is produced. It is misshapen, the surface is irregular, uneven, and covered with nodules about the size of a pea. The serosa is frequently thickened. Microscopically the marked increase of connective tissue is most conspicuous, and this, following the interlobular and intralobular vessels, by its contraction destroys the hepatic cells as well as the blood-vessels and bile capillaries. Frequently little remains of the hepatic parenchyma, and this is surrounded by wide connective tissue striæ. The arrangement of the hepatic lobules and of the hepatic trabeculæ is everywhere destroyed by the connective tissue which permeates it. In place of the lost liver cells we see occasionally a collection of brown pigment. Regenerative processes may also be observed in the shape of so-called newly formed biliary passages and blood-vessels in the newly formed connective tissue. The latter are not in communication with the portal vein, but with the hepatic artery. The newly formed connective tissue is usually fibrinous, and sometimes rich in cells, according to the age of the new formation. Sometimes the liver is smooth, in spite of considerable connective tissue proliferation in the organ, but only when the arrangement of this tissue is somewhat uniform. The cause of this difference is unknown.

Of those advanced forms which represent the terminal stage of a process existent for years, the earliest stages differ very materially. The organ is then uniformly enlarged, of greater consistence, its border dull, the surface

less smooth. Microscopically the previously described processes are present, but only to a slight extent.

By the pressure of connective tissue, many of the portal vein branches are destroyed; the consequence of this is stasis in the larger branches of the portal vein. The formation of new capillaries of the hepatic artery does not sufficiently compensate for this. Large branches of the portal vein may also be compressed by firm connective tissue proliferation.

The beginning of the process can scarcely be recognized macroscopically, and can only be fully determined by microscopic examination of the organ.

Fatty degeneration of the liver to a greater or less extent, associated with the extension of the process and the inconstancy of individual phenomena, furnishes very varying pictures, and presents an opportunity, if we have a tendency to schematism, to differentiate many dissimilar forms, for which, however, there is no necessity.

For instance, to mention but a few of these individual forms, we hear of monolobular and multilobular cirrhosis, according to whether few or many lobules have been embraced and enclosed by individual connective tissue proliferation. The cirrhosis is called bivenous when the connective tissue proliferation starts not only from the portal vein branches but also from the central veins.

Furthermore, we speak of a "cirrhose hypertrophique graisseuse," of a monocellular or intercellular form, of a "forme mixte," etc. Surre (31) differentiates eight forms of hepatic cirrhosis, and many authors consider this as by no means exhaustive.

The question has been mooted whether in the course of hepatic cirrhosis there is also a stage of hypertrophy of the organ. Great importance has been attached to the decision of this, especially in the discussion regarding the justification for separating hypertrophic hepatic cirrhosis as a special pathologic process. In this disease, which will be described minutely in the next chapter, a permanent and quite decided enlargement of the organ is especially characteristic.

Now there is no doubt that in the course of atrophic hepatic cirrhosis a periodical and more or less marked enlargement of the organ takes place. Any one who has seen many cases of hepatic cirrhosis will confirm this. This hypertrophic stage of the organ, besides being due to the hyperemia, the swelling of the cells, the increased and not yet contracted connective tissue, is also attributable to a more or less decided fatty infiltration of the cells. In atrophic hepatic cirrhosis, adenomatous neoplasms form which, perhaps, are later transformed into malignant tumors (carcinoma), and which naturally increase the circumference of the organ. Therefore, the causes of so-called hypertrophy of the liver in atrophic hepatic cirrhosis are many, and this alone, even when it persists for a long time, does not warrant us in assuming hypertrophic hepatic cirrhosis, and excluding Laennec's atrophic form.

SYMPTOMATOLOGY AND COURSE OF THE DISEASE

The disease certainly exists for a long time—occasionally for years—without giving rise to symptoms, and is often discovered while examining patients who come to us in consequence of some other affection. In keeping with the etiology, symptoms on the part of the gastrointestinal canal resembling those so frequently present in alcoholics are the first complaints of the patient. These consist of nausea, anorexia, eructations, vomiting, constipation, sometimes even diarrhea, pressure and a sensation of fulness in the epigastrium, even pains in this region, etc. The enlarged liver causes distress by pressure upon the neighboring organs. The patients are debilitated, the general condition suffers. Pain is rare in true atrophic hepatic cirrhosis. It occurs only when the inflammation especially attacks the capsule of the liver, and this is seldom. In the course of the disease irregular rises of temperature are occasionally noted, which come and go, and for which no cause can be assigned. Severe symptoms are observed only in the later stages, when disturbances appear which are due to the portal vein circulation. Mild jaundice may be present, but does not necessarily belong to the picture of atrophic hepatic cirrhosis. In consequence of the destruction of so many small portal vein vessels, there is stasis of the blood in the trunk of the portal vein which reacts upon the stomach and intestine. The consequence is an increase of the catarrhal symptoms in both organs and the appearance of ascites, which then becomes prominent in the picture of hepatic cirrhosis and continues permanently. Distention of the abdomen, and an increasing sense of weight and fulness in the same, are frequently the first symptoms which the patient observes. The ascites may develop to an extraordinary extent, and by forcing up the diaphragm as well as hindering respiration may cause the patient much distress. Frequently edema of the lower extremities follows. With the constantly increasing symptoms on the part of the gastrointestinal canal, the nourishment is deficient and the patients rapidly lose weight. The enormously distended abdomen forms a striking contrast with the body emaciated almost to a skeleton. Hemorrhages occur in consequence of decided stasis of blood in the esophagus, stomach, and intestines. These are occasionally only of the capillary form, so that hemorrhagic feces and the vomiting of black masses are observed. But these hemorrhages may not infrequently be also very copious, and threaten life; then large amounts of pure blood are vomited, and the dejecta are of a black, tarry color. At the autopsy ruptured varices are found to be the cause of this, especially in the lowest portions of the esophagus; but pure parenchymatous hemorrhages may also endanger life through their massiveness. The nutrition of the walls of the vessels also suffers and leads to hemorrhages in various organs, such as the skin, the mucous membrane, the retina, etc.; fatal epistaxis is also sometimes observed. The slight jaundice is partly

due to compression of the biliary channels in the liver and to biliary stasis due to the resorption of bile, in part it may be referred to duodenal catarrh.

The examination of the abdominal organs with an existing ascites is very difficult, and often is only possible after the fluid has been withdrawn. A hard, contracted, nodular liver is then usually felt; sometimes the organ is so small that it can no longer be palpated.

The condition of the spleen in hepatic cirrhosis is of importance. Frequently it is enlarged to two or three times its normal size; and this is a factor of great significance in the diagnosis of the affection, particularly in its last stages when the liver can no longer be palpated. Enlargement of the spleen is not only due to stasis of the blood in the portal vein, but to direct hyperplasia of its tissue, with an increase of the connective tissue. We must, therefore, assume that active processes are going on in the spleen, similar to those in the liver. Sometimes, however, splenic enlargement is absent, particularly if the capsule is tough and thickened.

Ascites.—Ascites requires a somewhat more detailed description. The fluid fluctuates in the abdominal cavity, and is to be attributed to the passage of fluid through the intestinal serosa into the peritoneal cavity. According to Quincke (32) some of the transudated fluid is poured out by the lymph-vessels of the diaphragm and of the parietal peritoneum, and this is independent of the portal vein circulation. When the ascitic fluid is withdrawn it is usually clear, amber-colored, with a specific gravity of 1.012 to 1.014, and contains from 0.6 per cent. to 1.2 per cent. of albumin. There is scarcely a trace of sugar or biliary coloring matter. Turbidity to any extent points to complicating inflammation of the peritoneum; blood may appear in small or in large quantities in consequence of stasis hyperemia of the serosa.

In addition to the previously mentioned stasis, dilatation of large venous areas which have a collateral relation to the portal vein, or may become thus related, plays an important part in hepatic cirrhosis. Notwithstanding the obstruction to the circulation, large quantities of blood from the portal vein system may be poured into the veins of the body and thus the portal system is greatly relieved. It appears, however, that the compensatory importance of this collateral development is greatly exaggerated; this value, at least clinically, is rarely observed.

Collateral Circulation.—1. The communication of the mesenteric veins with the veins of the abdominal walls. 2. The connection of the gastric veins with the inferior œsophageal veins and, through these, with the azygos vein. 3. The conjunction of the internal hemorrhoidal veins and the hypogastric veins. 4. The communication of the veins of Glisson's capsule with the veins of the diaphragm through perihepatitic adhesions. 5. A dilatation of the incompletely obliterated umbilical or paraumbilical veins which run along the ligamentum teres, from which point blood may

then be carried by the portal vein to the veins of the abdominal walls, particularly to the epigastric vein. These veins of the abdominal walls are more or less markedly dilated, and form a figure which bears a remote resemblance to *the head of Medusa* (*caput Medusæ*), and accordingly has received this name. The caput Medusæ, at least in its well developed form, is by no means frequent; we must not mistake for it simple dilatation of cutaneous veins, such as the superior and inferior epigastric veins combined, which represent nothing more than collateral tracts of the compressed inferior vena cava; these may be found in any form of abdominal dropsy, and not only when they are the consequence of an implication of the portal vein circulation.

Other and rarer collateral formations, for example, communication between the paraumbilical and the right iliac vein by a vein of the abdominal wall (Drummond), require no further mention.

The *pulse* is small and somewhat accelerated, but it is regular unless there are complications on the part of the heart.

The *respiration* is accelerated when ascites is present because the diaphragm is forced up, and its action is limited.

If death does not take place early from intercurrent diseases and complications, this is preceded by symptoms of increasing weakness and exhaustion. Not rarely the patient falls into a comatose condition with delirium, general convulsions, etc., and from this he never awakens. This gives us the impression that we are dealing with a condition resembling cholemia, such as is observed in long-continued stasis jaundice, or in acute yellow atrophy of the liver. As jaundice is absent or very slight in the affection in question, this cannot be attributed to intoxication with constituents of the bile, but it must be due to other products of metabolism which are not yet sufficiently known. The antitoxic function of the liver is unquestionably not its least important one—this we have previously referred to. But in this respect, as well as in many others, the organ cannot function on account of the constant and increasingly rapid disappearance of its parenchymatous cells. The toxins—probably entering from the intestine—penetrate the protective wall, flood the organism, and produce the condition which we designate as autointoxication. Perhaps the same substances, and not biliary constituents, cause the condition known as "cholemia" or "acholia."

Of the nature of these toxins we have, unfortunately, as yet no accurate knowledge, and in my opinion here, i. e., in the study of this toxin production, scientific investigators should lead the way. Up to the present time, we have considered only ammonia, the aromatic products (phenol, skatol, indol, etc.), the alkaloidal products (putrescin, cadaverin, neuridin, etc., which we have included under the name diamin, and a knowledge of which we mainly owe to the pioneer investigations of Brieger), and the products of metabolism of the bacteria from the intestine which have a

toxin-like effect, that is, in other words, the products of proteid decomposition.

In regard to ammonia, the investigations of Nencki, Pawlow, Hahn and others in dogs, with Eck's fistula (excluding the portal vein circulation) should be borne in mind. In their experiments upon animals, these investigators observed symptoms which closely resembled those of cholemia, particularly when nitrogenous food was administered.

Many authors (Heger (33), Schiff (34), Lautenbach (35) and others) found that the liver not only retains certain poisons which are introduced with the blood (nicotin, strychnin, hyoscyamin, etc.) but also destroys them or transforms them into non-toxic substances, or, at least, stores them up.

Special credit for working out and elucidating this question is due to Bouchard (36) and his pupil Roger (37). According to the latter the liver acts upon poisons in three ways: 1. It stores them (metallic poisons such as copper, iron, arsenic, etc.). 2. It excretes them with the bile (a rarer and less active process). 3. It renders the poisons more or less inert, i. e., destroys them or changes them into non-toxic combinations (vegetable alkaloids, peptones, toxic products of intestinal decomposition, ptomains).

This protective action of the liver, according to Roger, is due to the glycogen it contains, with which these poisons combine, and thus become non-toxic. It must be confessed that these teachings have not been generally accepted, and that they cannot as yet be regarded as incontrovertible. We shall see later, when discussing the treatment, particularly the dietetics of diseases of the liver, in how far we may utilize these views. Other experimental investigations to determine the principle on which is based this protective action of the liver toward poisons, I shall not discuss. They have given us no additional information.

Bouchard attempted to prove this important property of the liver indirectly by testing the toxicity of the urine in various diseases of the liver, and comparing it with the normal. He started with the hypothesis that the soluble poisons circulating in the body were excreted in the urine. If the liver had an antitoxic action, this must be limited in hepatic diseases, and, in consequence, the urine must be more toxic (the "urotoxic coefficient" increases). He found his deductions confirmed, and he drew from these many conclusions, some of which, however, are very questionable. If we accept all that has been stated, notwithstanding the criticisms at hand as to the results of experiments, we can no longer doubt the influence of the liver upon numerous poisons (accordingly upon those also which reach it from the intestine).

The investigations of metabolism in hepatic cirrhosis have not been very extensive, but, nevertheless, we have obtained some very remarkable results. In regard to the metabolism of nitrogen in hepatic cirrhosis,

Hallervorden (38) has occasionally found very large quantities of ammonia in the urine (up to 2.5 in the daily quantity). I also (39), and following me many other authors (Fawitzky (40), Gumlich (41), v. Noorden (42), Münzer (43), found the quantity of ammonia increased in comparison with the total amount of nitrogen (10 to 18 per cent. instead of the normal 2 to 5 per cent.), but the absolute figures very rarely reach the amount determined by Hallervorden (Mörner (44) and Sjöquist found in one case 2.4 grams). Yet from this alone we cannot determine a diminution of the urea-forming function of the liver, which, on the contrary, appears to be extraordinarily active, usually to the end. For, besides these large quantities of ammonia, large amounts of urea also are formed (30 to 40 grams in twenty-four hours). With the decreased excretion of urea which may be regarded as the consequence of a deficient ingestion of food, of inanition, the excretion of ammonia is also lessened. The relation of urea to the total amount of nitrogen of the urine is also but little altered (77 to 91 per cent. as compared with the normal of 80 to 90 per cent.). Some time ago I (45) expressed the conviction that other factors, and particularly an increased excretion of acid, caused an increased excretion of ammonia. In the urine I, as well as v. Noorden, occasionally found sarcolactic acid, as has lately Calabrese (46), while v. Jaksch demonstrated fatty acids, particularly acetic acid, valerianic acid, butyric acid and propionic acid. Weintraud (47) administered ammonia up to 9 grams in hepatic cirrhosis, and found no increased excretion of ammonia, but only the requisite and expected increased excretion of urea. Therefore, if urea is produced in the liver alone and not in other organs, which, however, is unlikely, we must not conclude from these findings that this important function is sufficiently performed by the remaining parts of the hepatic tissue, and that life ceases before the disease leads to marked and distinctly recognizable anomalies in urea formation. The recent conclusions of Schittenhelm (*Deutsches Arch. f. klin. Medicin*, LXXVII, H. 5 u. 6) in the main agree with my views; he also noted an increase of the excretion of ammonia in the urine in destructive hepatic affections, and refers this to the acidity of the organism. Peptone is positively absent from the urine in hepatic cirrhosis; albumoses are occasionally found, but they are rare and in insignificant amounts. Uric acid excretion is within normal limits, as well as the alloxur bodies (prior stages of uric acid) which have lately come within the circle of scientific investigation. The finding of leucin and tyrosin is more than questionable. von Jaksch (48), who has made very extensive investigations in regard to the nitrogen excreted in the urine in hepatic diseases, has come to the conclusion that, in the main, it appears as urea, and that other nitrogenous products are not found in increased amount.

The heat-generating function of the liver must naturally suffer as soon as the activity of the organ is decreased. As, however, the normal function

of this organ in proportion to the heat-producing power of the entire organism is not definitely known, in pathologic processes, naturally, little can be said on this point. I believe that this function of the most important glandular organ of the body is not insignificant. However, there is as yet no possibility of our determining the effect of absence of its function in the human organism.

Sugar is sometimes found in the urine of these patients. Yet, on account of the rarity of the finding, I do not regard it as in organic relation to the underlying affection (hepatic cirrhosis), but merely as an accidental complication.

Steinhaus (49) found in the majority of cases of hepatic cirrhosis investigated by him (in 11 out of 12) distinct connective tissue proliferation of the pancreas resembling that of the liver, and he believes that occasional diabetes in such patients may be attributed to this condition. These observations, therefore, coincide with the opinion expressed above that diabetes is here not directly due to the hepatic disease.

Alimentary glycosuria has also been observed, but too rarely for us to deduce diagnostic conclusions therefrom, as do the French authors. This is true, at least, regarding grape-sugar (intake and excretion). With the intake of levulose, the condition is different (H. Strauss (50), Lépine (51)), for it has been observed that patients with disease of the liver usually develop levulosuria in contrast with normal persons. In 26 of 29 patients examined by Strauss, this levulosuria could be demonstrated.

As, however, according to the reports of Landsberger (*Deutsche med. Wochenschr.*, 1903, Nr. 32), similar conditions are not infrequent in healthy persons, our anticipation of having found an important aid for the diagnosis of alimentary levulosuria has met with disappointment.

COMPLICATIONS

Alcohol, which is the chief cause of hepatic cirrhosis, also damages a number of other organs, such as the heart, kidneys, vessels, etc. Combined with hepatic cirrhosis we also frequently find myocarditis, nephritis, and arteriosclerosis, but rarely pachymeningitis or chronic meningitis. The other complications of hepatic cirrhosis, such as portal vein thrombosis and fatty liver, have already been mentioned. Myocarditis with its sequels, edema, congestion of the liver, etc., as well as nephritis, is often the cause of the fatal termination of our cases.

The not infrequent combination of hepatic cirrhosis and primary carcinoma of the liver is remarkable, as is the connection between hepatic cirrhosis and peritoneal tuberculosis which has been referred to previously.

DIAGNOSIS AND PROGNOSIS

Easy as the diagnosis of hepatic cirrhosis may be in many cases, in others it may be most obscure. In alcoholics with enlarged hard liver, recognizable enlargement of the spleen, and moderate ascites, the diagnosis presents no special difficulty. This only becomes perplexing when we are dealing with patients in whom marked ascites makes palpation of the abdominal organs impossible. Then we must first determine whether a general circulatory disturbance is the cause of the ascites. If the heart, lungs and kidneys are normal, if there is no general edema, it is exceedingly likely that there is a circulatory disturbance in the course of the portal vein, and, when combined with chronic alcoholism, hepatic cirrhosis may be presumed to be the cause. But only a probable diagnosis can be made in such cases. For portal vein stasis may be due to many other causes (compression of the portal vein by tumor, thrombosis of the portal vein, carcinoma of the peritoneum, of the omentum, etc.). When the ascitic fluid is removed, and we are able accurately to investigate the abdominal organs, we frequently arrive at a correct diagnosis.

The differential diagnosis between hepatic cirrhosis and chronic, as well as tuberculous, peritonitis is especially perplexing. The latter affection combined with hepatic cirrhosis is not so rare as was formerly supposed. The proof of tuberculosis in other organs, the character of the ascites, which in peritonitis is frequently hemorrhagic and also shows a higher specific gravity with a greater amount of albumin, will in many cases lead to a correct conclusion, but we must admit that errors in diagnosis cannot always be avoided. The existence of a chronic perihepatitis, perihepatitis chronica hyperplastica, must also be considered in the differential diagnosis. The great thickening of the capsule leads to compression of the organ, to disturbance of the portal vein circulation, and to destruction of the hepatic parenchyma. The organ, in consequence of the marked contraction of its capsule, becomes small, atrophic, hard, and ascites appears; in brief, a picture which is very difficult to differentiate from atrophic hepatic cirrhosis. A similar affection is described by Pick (*Zeitschr. f. klinische Medicin*, Bd. 29, Heft 5 u. 6), pericardial pseudo-hepatic cirrhosis, i. e., perihepatitis chronica hyperplastica combined with chronic pericarditis and obliteration of the pericardium.

Duration.—The *duration of the disease* cannot be determined with certainty, since, on account of the mild symptoms, the time of onset is not known. In this respect, therefore, individual authors express very varying opinions, and they estimate the duration of life at between one and ten years. The earliest possible recognition of the disease, the whole state of the patient, his circumstances, the avoidance of everything injurious, and, particularly, abstention from alcohol, etc.—all, naturally, have an important bearing upon the course of the disease.

Prognosis.—As to the prognosis in a positive case of atrophic hepatic cirrhosis, all authors are unanimous in declaring the disease to be fatal. Yet many cases of recovery, even in advanced hepatic cirrhosis with ascites, have been reported (I shall only mention Liebermeister, Rosenstein, Semmola, Leichtenstern). Naunyn especially emphasizes that, in his experience, the cure of atrophic hepatic cirrhosis is sometimes spontaneous. Ehret, upon the basis of one of his own cases, has recently expressed himself on this point most explicitly, and entirely agrees with Naunyn. I have never seen an actual cure of atrophic hepatic cirrhosis. Where this appeared to be the case, there was some doubt as to the diagnosis. Syphilitic hepatic cirrhosis will be described separately.

TREATMENT

The treatment is, in the main, symptomatic. The removal of the etiologic cause, the early and entire withdrawal of alcohol, and the forbidding of pungent spices are most important; in malaria we employ quinin, in syphilis potassium iodid, and even mercury given cautiously. Where syphilis cannot positively be determined, potassium iodid should be given a trial. The patients should not be exposed to the deleterious action of lead, arsenic or phosphorus. As soon as the pressure of ascites causes distress, paracentesis abdominis is indicated; early puncture, however, is not advised; the value of this is very problematical. The first deposits of the newly formed ascitic fluid are always most rapidly excreted. When the pressure of fluid in the abdomen has become distinctly positive, accumulation takes place more slowly. By early and, consequently, frequent puncture, a large quantity of albumin is withdrawn from the body, and this hastens the destruction of the patient. In contrast with this is the value of early puncture in decreasing venous stasis in the abdominal organs; this is supposed to hasten the establishment of the collateral circulation between the portal vein and the organs of the body, etc., which is so desirable to the patient, but is as yet by no means proven. In regard to the necessity of tapping, I determine this from the symptoms of the patient, and, since general rules cannot be given, I can only advise it. In some patients we must perform this little operation early and often; in others enormous quantities of fluid in the abdomen occasion no special symptoms.

When the patients complain much of abdominal pressure, and the cardiac activity and respiration are implicated by the displacement of the diaphragm, aspiration must be performed. Not only these difficulties, but many others, then seem to disappear as if by magic. The appetite is stimulated, a sufficiency of food is ingested, the bowels become regular, and diuresis improves. Aspiration is often followed by an amelioration of the entire condition which lasts for some time.

In atrophic nutmeg liver due to diseases of the heart, lungs, and kidneys,

the treatment of the underlying affection, particularly of the heart, must be our first object. Digitalis is then the most valuable remedy, and we must attempt by diuretics to prevent the accumulation of fluid or to remove that which has been formed. Cream of tartar, potassium acetate (always in large doses, 10, 15 to 20 grams a day), juniper, and special diuretics such as boneset tea (50 grams macerated, placed in a liter of cold water and allowed to boil for three or four hours, then poured off and filtered; always to be freshly made, and the entire amount taken in twenty-four hours). Urea, which was recently strongly advised as a diuretic by Klemperer (10 to 20 grams a day), I have not found specially valuable. In my experience it acts no better than the remedies mentioned above when given in the same quantity, and these may be prescribed when the kidneys are normal without damaging these organs (in the diseased kidney urea is even without effect and, possibly, harmful). In England balsam and resin of copaiba are much employed (1 or 2 grams daily in capsules). Potent diuretics which enjoy a reputation in hepatic cirrhosis are diuretin (4 to 6 grams daily), agurin (1 gram three or four times daily), theophyllin (0.2 to 0.3 gram three or four times daily), theocin, the double salt of caffein (from four to six powders daily, each 0.2 gram), and, above all, calomel. This last remedy calls for a more minute description. I advise its employment only when the kidneys are normal. Then 0.2 of a gram of calomel are given three times daily for three days, followed by a pause of several days. The oral cavity must be carefully watched. With marked diarrhea, opium should be given at the same time. I am afraid of calomel, which occasionally gives such excellent results, because the stomatitis which, notwithstanding the greatest care, sometimes follows, may debilitate the patient extremely. Its specific action upon the process in the liver, which is presumed by some authorities, has been nowise proven. Perhaps smaller doses (0.02 to 0.06, five or six times daily), which are specially advised by Saccharjin in hypertrophic hepatic cirrhosis, would also be of value here.

Spa treatment of the affection under discussion at Carlsbad, Marienbad and Kissingen, etc., is highly recommended, and sometimes also the employment of artificial Carlsbad salt. But a specially beneficial action upon the liver and the processes going on there these salts cannot have; even when given by the mouth (54) in large doses they do not enter the bile. Hence the value of alkalies can only lie in their favorable effect upon the gastrointestinal catarrh. Symptoms referable to the gastrointestinal tract are often very prominent in these patients, and primarily demand treatment, which, unfortunately, is often without result. Sometimes these symptoms point unmistakably to gastric catarrh (pain in the epigastrium, anorexia, nausea, vomiting), at other times to involvement of the intestine which is usually manifested by uncontrollable diarrhea, more rarely by obstinate constipation. Disturbance of the circulation of the blood in the

portal vessels is certainly the cause of these conditions. In this case the symptoms of gastrointestinal catarrh, which are prominent, call for relief. Here I must refer to the special treatment of these affections, and shall only mention that the spa treatment is very beneficial (besides the springs mentioned above also the waters of Vichy, Neuenahr, Ems, Marienbad, Kissingen, etc., as well as the salt springs of Homburg, Wiesbaden, etc.), especially when constipation and meteorism exist. The stagnant, decomposing masses are discharged, and absorption of the freshly digested foods is promoted, hence the action of the toxic, deleterious products of intestinal bacteria (toxins, ptomains) is limited. In regard to the deficient antitoxic function of the liver, the French (Bouchard, Roger, Dujardin-Beaumetz (55), and others) assign the first place in treatment to "intestinal antisepsis." Charcoal, bismuth salicylate, salol, resorcin, naphthalin, sodium salicylate or sodium benzoate, etc., hydrochloric acid (with subacidity of the stomach), have all been warmly advocated by them, and are frequently employed combined with a diet which will soon be described in detail. So far as we know, antisepsis is carried out in the stomach and the upper portions of the intestine by hydrochloric acid; in the lower portions of the small intestine possibly (?) by the bile acids. It is well known that in advanced hepatic cirrhosis, even when there is little jaundice, only small quantities of bile flow into the intestine, consequently only small amounts of biliary acids find passage there. Connective tissue proliferation in the liver may be enormous, and the greater portion of the hepatic cells may be destroyed, so that microscopically but scant remains of these, which, apparently, are in great part atrophic, are to be found. Deficiency in bile formation and the resulting incomplete antisepsis in the intestine become more pronounced, and the antitoxic property of the liver, on account of the few undamaged hepatic cells which remain, is exerted insufficiently. In the course of hepatic cirrhosis we by no means rarely meet with conditions which point to autointoxication of the organism with these intestinal toxins. These conditions have already been explicitly described (56).

In Germany, quite correctly, the theory of "intestinal antisepsis" has met with little acceptance. The extremely artificial superstructure erected in France lacks a sufficiently firm foundation, the entrance is not free from danger, and it may very readily and suddenly collapse. Preliminary studies upon which to found this treatment must be characterized as entirely inadequate. I am convinced that, in the disease in question, as little headway has been made with intestinal antisepsis as, for example, in enteric fever, which in France is treated according to similar principles. The administration of small doses of antiseptics is useless, large doses are decidedly dangerous. A combination of various antiseptic remedies (of each almost its toxic dose) has given no better results. We will do well to continue to be skeptical in regard to this therapy, and to rely upon purgatives

such as aloes, senna, calomel, rhubarb (Heim's pills have been most extensively used in the treatment of hepatic cirrhosis), and the laxative bitter waters. It is simply impossible to disinfect for years the intestine of a human being, all the more so since we do not know which bacteria should be destroyed, and which toxins should be removed. Continued research is here necessary, and then perhaps, but only perhaps, treatment of this kind may be successful.

A chronic disease, the course of which, like the one in question, is so prolonged, naturally necessitates care in the diet, and here the patient's food must be simple and non-irritating with a sufficient amount of calories. It should be composed of milk, carbohydrates in the form of wheat bread, crackers, flour, zwieback, mashed potatoes, cocoa, and rice, with the addition of butter and meat. Only the more easily digested and nutritious vegetables, such as green peas, turnips, asparagus, cauliflower, and spinach, are to be allowed, while, on the other hand, those which cause distention—such as all kinds of cabbage—are to be avoided.

As an ideal diet in hepatic cirrhosis the French have praised and practically employed one strictly of vegetables. The guiding thought is this—that in its employment the formation of toxins (from meat) is suppressed, and intestinal antisepsis is facilitated. In carrying out such dietetic rules, the utmost care is necessary. Few patients with hepatic cirrhosis can adhere to such a diet on account of their especial tendency to gastric and intestinal ailments. For individual points in the diet, I must refer to authoritative text-books (57).

Surgical Measures.—To facilitate the establishment of the above mentioned collateral circulation between the portal vein and the veins of the body, that is, to make it possible, and thus relieve the dangerous consequences of congestion in the portal system, efforts have been made, following Talma's (57 a) advice, to fasten the omentum, and also the spleen, to the abdominal wall. In this manner adhesions containing new vessels are formed, and may bring about the desired condition. This operation is not always successful; disappointment and even serious results (gangrene of the omentum, infections) have followed; but, on the other hand, sometimes results which are favorable. In some cases, after the operation, the ascites was checked, and the patients left the hospital in the best condition and able to carry on their occupations (Talma, Drummond and Morison, Neumann (57 b)). Such favorable results as these from Talma's operation justify us, in suitable cases, in resorting to it. With our increasing experience regarding the operation itself, and in the choice of suitable cases, we can hardly doubt that in the future these results will be even more gratifying. Experimental investigations (Tillmann (57 c)) confirm the practicability and the benefits of Talma's operation; comprehensive reviews by Friedmann (57 d) and Rohmer (57 e) give critical reports of numerous cases operated upon, and a recent publication by Lenzmann

may be referred to (*Deutsche med. Wochenschr.*, 1903, Nr. 48, p. 843) in which suggestions have been given regarding the choice of cases to be operated upon.

C. HYPERTROPHIC HEPATIC CIRRHOSIS

PATHOLOGICAL ANATOMY

In important points this affection differs pathologico-anatomically as well as clinically from atrophic hepatic cirrhosis, and therefore, as is now generally admitted, is entitled to a special position.

Up to the time of death the liver usually remains greatly enlarged. The surface is tumid, it is of firm, elastic consistence, its color strongly icteroid, yellow to green. Upon section a decided increase of connective tissue is seen; this is not only found in the interlobular spaces, but also in the hepatic lobules themselves. In spite of the enormous increase of the connective tissue, the mass of the hepatic parenchyma is unaltered. The connective tissue does not contract, the liver cells are not destroyed; on the contrary, according to some authors (Ackermann (58), Aufrecht) they are enlarged, hypertrophic, and mostly polynuclear. That the connective tissue proliferation in hypertrophic hepatic cirrhosis takes place especially around the biliary passages and, according to the view of the French, chiefly Charcot and his pupils, originates from these and not from the blood-vessels, has by no means been proven, and has been denied by me for many years (*Verhandlungen des Congresses für innere Medicin*, 1892).

Since the finer biliary passages and the blood-vessels coalesce in the same interstitia, it is hardly possible from the results of microscopic investigation to maintain with any degree of positiveness that the one system, and not the other, is implicated in the connective tissue increase. This is not contradicted by the fact that, in many areas, connective tissue proliferation is greatly developed, particularly around the biliary passages, that their walls are thickened while the lumen remains open. The biliary channels, on account of their structure, form a prominent part of the connective tissue masses. The incorrect view of the French is, as was previously remarked, based upon the fact that they do not distinguish "biliary cirrhosis," which we shall specially describe later, from hypertrophic cirrhosis, and this has caused much confusion. So-called, newly formed biliary passages are more numerous than in the atrophic form. It must be expressly stated that the differences between these two forms are only marked when we compare well-developed classical cases, that these, however, are by no means always typical, and that these forms merge into one another. Thus, in hypertrophic hepatic cirrhosis there is sometimes contraction, atrophy of the cells, etc. The French have easily surmounted

these difficulties by creating their "forme mixte." But this, at the same time, indicates that special differences between atrophic and hypertrophic hepatic cirrhosis do not exist. The pathologic anatomy of the disease, which apparently occurs in France much more frequently than in Germany, has been particularly studied by the French—I mention only such names as Charcot (59), Ollivier (60), Hayem (61), Gombault (62), Hanot (63), Schaehmann (64) and, among German authors, besides Aekermann (*l. c.*), Rosenstein (65) and Stadelmann (66). In France this form of hepatic cirrhosis is named after the most eminent authority in the realm of this disease, Hanot's disease. After all is said, we must frankly admit that the true cause of differences, even of a pathologico-anatomical nature, between atrophic and hypertrophie hepatic cirrhosis is unknown. It is worthy of mention that, occasionally, even well-developed cases of hypertrophic hepatic cirrhosis cannot be recognized macroscopically, but are only determined by microscopic examination. This is due to the peculiar, soft, but only slightly prominent, structure of the richly developed connective tissue, and the absence of contraction.

SYMPTOMATOLOGY

The onset of the disease, like atrophic hepatic cirrhosis, is accompanied by indefinite symptoms on the part of the gastrointestinal tract. The patient complains of anorexia, vomiting, pressure in the epigastrium, constipation, and diarrhea. A very intense jaundice soon appears, so that this form of the affection has been designated in France as "cirrhose hypertrophique avec ictère." The liver is greatly enlarged, is coarse and hard, with a rounded margin, and remains enlarged to the end. The jaundice may disappear in the early stages of the disease but may several times recur in paroxysms until it finally becomes permanent. Besides enlargement of the liver, we usually find a very marked enlargement of the spleen. Ascites is almost invariably absent. There is a marked tendency to hemorrhage. In spite of the jaundice, the feces usually show bile. Death occurs from exhaustion after a prolonged duration of the disease, which runs a longer course than atrophic hepatic cirrhosis (four, five, ten to twelve years), and is frequently preceded by the symptoms of autointoxication and by cerebral phenomena. Irregular rises in temperature are not rare. The cause of the enlargement of the spleen is doubtful; it cannot be congestion, as in atrophic hepatic cirrhosis, for this is entirely absent from the clinical picture. Nothing remains but the assumption of a process in the spleen resembling that taking place in the liver. Rosenstein has mentioned a decrease of the red blood-corpuscles.

Disease of the kidney is rarer than in atrophic cirrhosis.

As *complications* there are to be mentioned general and local peritonitis (perihepatitis, perisplenitis), as well as myocarditis.

ETIOLOGY

Hypertrophic hepatic eirrhosis, similar to the atrophic form, oceurs ehiefly in men, less frequently in women. It is quite a rare disease, and is apparently restrieted to certain loealities. Thus, in France and Holland (Rosenstein) it is eomparatively frequent, while in Germany, at least in its typical forms, it is very rare. Most authors attribute the affeetion to alcohol, others contradiet this (Rosenstein); it is eertainly very remarkable that, notwithstanding the plentiful ingestion of alcohol in Germany, the affection is there so rare, while, in other regions, in spite of the same conditions, the disease is often noted. This forces us to the conelusion that alcohol cannot be the only eause, but that an unknown faetor is added. It has also been assumed that it is due to a parasitie disease of the biliary ehannels (baeteria, protozoa). Positive proof of this is laeking.

DIAGNOSIS AND PROGNOSIS

In the stage of onset the affection is just as diffieult to reeognize as atrophie hepatic eirrhosis. The gastrointestinal symptoms are not charaeteristic, the jaundice may be purely catarrhal, and is the more likely to be considered such beeause this symptom, as well as the enlargement of the liver, often decreases. Only with the permanent enlargement of the liver, the decided splenic tumor, the persistent jaundice, does the diagnosis, until then tentative, beeome positive.

Prognosis.—The prognosis is unfavorable. Recovery from the well-developed disease is unknown; that the malady may be arrested, or even cured, in the stage of onset by suitable treatment is possible, but not proven, since, in this stage, the diagnosis is still uncertain. It is at least a source of comfort to know that the course of the affection may be very prolonged without decidedly interfering with the aetivity of the patient.

TREATMENT

At the onset of the malady the treatment usually employed in gastro-intestinal affections is to be instituted; the jaundice present, which is generally regarded as catarrhal jaundice, should receive appropriate—also dietetic—treatment. The effeets of the abuse of alcohol must be combated, and its future avoidance always be insisted upon by the physician.

In the case of hypertrophic hepatic eirrhosis the employment of calomel in fractional doses, as advised by Saccharjin (0.02–0.05, four or five times daily), is particularly valuable. According to this author, the drug has a specific action and brings about a eure. But that a specific action upon the process in the liver is not produced by the use of calomel, and that the expected result frequently does not follow, must be especially emphasized. Nevertheless, with all necessary precautions (thorough care of the

mouth, calomel for three or four days, then a pause of three or four days, then another calomel period, etc., the treatment to be kept up for about four weeks), the calomel treatment is advisable. Potassium iodid should also be tried in hypertrophic hepatic cirrhosis, although I have seen little success from its employment, nor have I found such reported in literature. Ebstein (66) maintains that he has had good results with potassium iodid.

The disturbances of metabolism in the hypertrophic form do not differ from those in the atrophic. Absorption and the assimilation of food show no deviation except that, perhaps, on account of the deficiency of bile in the feces and the jaundice present, the absorption and assimilation of fats in the intestine are disturbed. The foundation for nutrition is milk, to which carbohydrates and proteids should be added as required. Details may be found in text-books on dietetics (52).

D. BILIARY HEPATIC CIRRHOSIS

In France the disease under discussion is usually grouped with hypertrophic hepatic cirrhosis, as has already been mentioned, and this has caused great confusion. We consider it advisable sharply to differentiate the two conditions, and in Germany, so far as I can see, we designate by this term the cirrhosis which arises in connection with mechanical closure of the bile channels, especially the common bile duct, either by stone, by a tumor, by adhesions, by cicatrices, etc.

ETIOLOGY AND PATHOLOGY

After prolonged biliary stasis (Janowski) necrotic foci due to destroyed hepatic cells are found in the liver. These are absorbed, small cell infiltration appears about the surrounding tissue, and, later, connective tissue proliferation develops. The bile channels are also thickened by infiltration and connective tissue formation. New bile channels are formed in the same manner as in ordinary hepatic cirrhosis. The connective tissue increase, as well as the atrophy of the parenchymatous cells, may finally attain the extreme proportions seen in atrophic hepatic cirrhosis. Consequently, as in the other affection, the picture of portal vein stasis appears, and in this stage biliary cirrhosis can no longer be differentiated from cirrhosis due to other causes (Litten (67), Mangelsdorf (68)). We then find fibrinous connective tissue bands which constrict and permeate the acini, connective tissue proliferation around the portal vein branches which become thickened and impermeable, and newly formed biliary channels, etc.

Experimental investigation, in the main, agrees with observations in man, but the consequences of experimental biliary stasis are much less prominent in the liver. Infection with its consequences (purulent inflammation, abscess) may complicate the situation in the animal experiment, and it is not always absent in man.

SYMPTOMS

The affection runs its course under the picture of severe jaundice, of cholemia, that is, of autointoxication, and usually with such rapidity that the severe hepatic changes above described do not have time to develop completely. The disease rarely lasts longer than two years, and, by its more rapid course, as well as by the absence of decided enlargement of the spleen, biliary hepatic cirrhosis differs from hypertrophic hepatic cirrhosis to which it otherwise presents great similarity, especially if the liver is greatly enlarged, which is often the case. In the differential diagnosis the observation of the feces is important. In hypertrophic hepatic cirrhosis they are more or less bile stained; but in biliary cirrhosis they are acholic, and in the latter affection the icterus is usually more intense. Ascites, as well as the other signs of portal vein stasis (also enlargement of the spleen), may develop in the terminal period (Janowski (69)).

TREATMENT

The treatment depends, in the main, upon the etiology. As the affection always depends upon mechanical causes which cannot be removed by internal treatment, in biliary cirrhosis we are therefore compelled to resort to surgical measures. Stones must be removed by operation, and, in the case of cicatricial adhesions and tumors which cannot be removed, cholecystoenterostomy is to be performed, that is, the gall-bladder is to be united with the intestine. This operation seems to be too rarely performed, and we cannot sufficiently emphasize that it is calculated to save the life of many a patient, or, at least, decidedly to prolong it, and gives us fresh hope.

The greatest attention must be paid to the nutrition, and, in the main, the same laws are operative as in hypertrophic hepatic cirrhosis. Carbohydrates and proteids are preferable, while the fats should be as far as possible excluded, since, in acholia of the intestine, fats are insufficiently absorbed; on the other hand, milk should be employed to the fullest extent.

E. SYPHILIS OF THE LIVER

That syphilis of the liver produces interstitial hepatitis may be regarded as certain; nevertheless, one and the same poison, as must be admitted, may produce such different forms of disease of the liver that we cannot regard it as strange when we see the same conditions as in the case of alcohol.

The connection between syphilis and hepatic cirrhosis is not always easy to recognize, and often can only be determined late, provided the history of our patients does not furnish the clue; for, clinically, the stage of onset of syphilitic hepatic cirrhosis very rarely differs from that of the

alcoholic form. In syphilitic hepatic cirrhosis the development of connective tissue rich in cells takes place, and this has a tendency to marked cicatricial contraction, so that the tissue later becomes tough and fibrous, and the cellular elements disappear. The parenchyma of the liver is also destroyed, and fatty degeneration and atrophy occur in the hepatic cells. It is quite necessary, too, to differentiate congenital syphilitic hepatic cirrhosis from acquired hepatic syphilis.

The first is found in children with congenital syphilis, and here the above mentioned characteristics of *diffuse* hepatic cirrhosis are most evident. The liver is distinctly enlarged, tough, brownish, with many signs of new and old connective tissue in the interlobular structure, and this may also permeate the lobules. The blood-vessels are greatly thickened, their walls show cellular infiltration. But, besides the diffuse connective tissue increase which presents the greatest similarity to that of ordinary hepatic cirrhosis, there are massive connective tissue strands in individual areas and foci with an accumulation of small cells.

In such children the liver is usually hypertrophic, probably because these children early succumb to the underlying affection (syphilis and its consequences), that is, before the atrophy which is observed in some cases that live longer has time to develop. The liver is sometimes smooth, sometimes flabby and notched, sometimes also coarse and granular. Besides the diffuse disease of the organ, we find—however, more rarely—a formation of gummata in the form of small nodules distributed over the entire surface. The process in the liver strongly resembles syphilitic disease of other organs. The disease without doubt begins during the fetal period; the fetus is often decomposed, or the infants die a few weeks or months after birth. Syphilis of the liver is the most frequent phase of hereditary syphilis.

Hepatic syphilis of adults is a late phenomenon of acquired syphilis appearing in the tertiary stage, and may be divided into two different forms, which, however, may also be combined.

1. The diffuse form corresponds to syphilitic cirrhosis of children and to alcoholic cirrhosis, and neither pathologico-anatomically nor clinically can it be positively distinguished from the latter. Syphilis and alcohol are usually combined as deleterious factors in one and the same patient.

2. Syphiloma of the liver, that is, gummatous hepatitis. Gummatous nodules are characteristic of this form; they vary in size from that of a nut to that of an apple, are mostly found upon the concave surface of the liver near the suspensory ligament, but may also be noted at the porta hepatis in Glisson's capsule. These nodules show a tendency to caseation, to decay, and, in connection with this, to contraction. Then deep furrows develop and cause constriction of the liver, hence the organ may be separated into individual lobes ("lobulated liver"); these constrictions consist of hard, tough connective tissue, perhaps also of remains of a caseated

gummatous tissue, and sometimes they contain unchanged gummatous nodules. Upon the smaller and larger blood-vessels (hepatic arteries, portal vein) connective tissue proliferation is frequently found in the form of syphilitic endarteritis. The serosa of the liver is usually thickened in consequence of inflammatory processes which may also cause extensive adhesions to the neighboring organs.

Finally, in hepatic syphilis of adults, as in that of children, the syphilomata may be of such a miliary form that a certain resemblance to tubercle is noted. In adults gumma formation, and in children the diffuse development of connective tissue, is typical of syphilis of the liver. But in adults other symptoms of tertiary syphilis are usually present (testicles, meninges, bones, mucous membranes, etc.).

SYMPTOMS AND CLINICAL COURSE

Syphilitic hepatic cirrhosis, at least so far as the gummata are concerned, usually runs its course without symptoms, and is frequently discovered only at autopsy. In inflammation of the serosa, if adhesions form, pain in the hepatic region is not a rare complaint. Frequently, however, the cicatricial retractions, the lobulation of the liver, as well as the large gummata, may be recognized at the examination, but the latter by their great nodular prominences often lead to errors in diagnosis by being mistaken for malignant tumors.

If the gummata and the cicatrization are situated at the porta hepatis, and form a constriction in this area, the well known symptoms of stasis, even icterus gravis, may arise in consequence of closure of the common bile duct.

[Gummatous hepatitis frequently produces an irregular type of fever. This is sometimes manifested by chills, fever and sweats occurring at irregular intervals and at other times by a moderate fever of intermittent or remittent type without chill. Frequently the liver is palpable, its consistency increased, and it is tender to the palpating hand during the febrile attacks. Rest without medication results in relief in some instances. The recognition of the cause with appropriate treatment with mercury and iodids is followed by entire relief.—Ed.]

As the liver is otherwise well able to perform its functions, and diffuse processes are rare in the adult, as has been mentioned, the constitutional condition is in most cases not affected, nor is life endangered, by the disease. Only when diffuse syphilitic hepatitis develops is the entire clinical picture of atrophic hepatic cirrhosis presented.

Amelioration and arrest of the affection, particularly under proper treatment, is very frequent.

Amyloid degeneration of the liver, spleen, and kidneys may follow, as in any cachexia; in severe syphilis this is not unusual, and it may then both complicate and disguise the clinical picture.

In children, usually in the new-born, the inverse is the case. The children are atrophic, cachectic, and do not thrive; ascites, enlargement and all of the other symptoms of diffuse hepatitis develop rapidly, and the children, as a rule, soon succumb to the disease. In milder cases, however, the affection may last for years. Amyloid degeneration of the liver and of other organs is then a frequent complication. The children, even the mild cases, rarely reach puberty, they have no vigor, they develop slowly, and remain weak and thin—just as is commonly observed in syphilis hereditaria tarda.

DIAGNOSIS

The diagnosis of syphilitic disease of the liver in young children is usually easy. The history of the parents, the presence of other symptoms of hereditary syphilis, and the examination of the children, soon make the diagnosis positive. The distended abdomen, the enlarged, hard liver which is readily palpable, the large spleen, the symptoms of congestion, ascites, etc., and the absence of other etiology pointing to hepatic cirrhosis, reveal the true nature of the malady even when the parents falsify their history during the examination, and conceal their own syphilis.

In adults, confusion with alcoholic cirrhosis on the one hand, and malignant tumors of the liver on the other hand, is easily possible. Even without a history, other signs of tertiary syphilis, such as cutaneous ulceration, enlargement of the bones, and ulceration in the throat, point with certainty to the correct conclusion. A complicating amyloid degeneration should be remembered, but should not lead us astray. If there is no other reason for its existence, in doubtful cases it favors syphilis. In a differentio-diagnostic respect, the slight cachexia may be considered as against carcinoma.

As contra-indications of alcoholic cirrhosis there are varying symptoms —longer-continued, spontaneous improvement, the enormous spleen, the size of the nodules, the marked globulation of the liver, and the possible absence of alcoholism, etc.

Confusion with simple corset lobe liver is easily possible. But this almost always occurs in women, and the furrow from lacing is a single one, although it may become very deep and almost divide the liver into two parts, while in syphilitic hepatitis there are several cicatricial bands, therefore lobulation occurs in several areas of the organ.

TREATMENT

With early and proper specific treatment, the disease may be arrested, the process retarded, and even a relative cure take place. The etiology and an early diagnosis are, therefore, of vital importance for the patient. Cicatricial constriction can never be removed, and, according to our present

experienee, it may be regarded as more than doubtful that a well-developed diffuse proeess ean be eured or even arrested.

Nevertheless, when syphilis has been determined and disease of the liver has appeared, a eareful antisyphilitie treatment with potassium iodid and mercury is not only invariably advisable but direetly indieated, and this treatment should be instituted even when the diagnosis of syphilitie hepatie disease is doubtful. Frequently, as is not unusual in syphilis, the result of treatment eonfirms the diagnosis. Cautious treatment with potassium iodid and mereury is not injurious if the patient's kidneys are normal and he is eontinuously under professional obsevation. The physieian should, therefore, first try potassium iodid or sodium iodid in inereasing doses, so that after a time several grams of the iodin salt will be given daily, and in eombination with this, as soon as we see that the iodin has had its effeet and the eondition of the patient will permit it, treatment with mereury should be begun (preferably a thorough inunction eure). Deeided pain in the hepatie region is the consequence of perihepatitis, and may justify sueh proeedures.

The other symptoms of hepatie disease are to be treated in aeeordance with the method established for the treatment of atrophic and hypertrophie hepatie eirrhosis.

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NEOPLASMS OF THE LIVER AND BILIARY PASSAGES

By FR. KRAUS, Graz

The development of the surgery of the liver and gall-bladder in the last few decades, gradual at first but lately proceeding with more rapid strides, has steadily intensified the practical interest of the profession in neoplasms of the liver. There are surgeons who dream of the complete eradication and actual cure of these diseases, even of malignant growths, by the aid of a diagnostic exploratory incision. Even more may, perhaps, be expected on account of the positive etiologic relation of carefully chosen cases of carcinoma of the biliary passages (perhaps also of the liver) to cholelithiasis if the operative treatment of gall-stone disease is resorted to early enough. If, however, the knife can actually aid us, and to avert the dangers in general of cholelithiasis, of which lithogenous cancer is only *one,* a very limited but, as I believe, sufficient experience has taught us that in permanent jaundice which is the result of an already present carcinoma of the common bile duct, operative interference is of comparatively little use, and even exploratory laparotomy, in individuals whose common bile duct is occluded by a malignant tumor, is very badly borne. The danger involved, therefore, in the operation for gall-stones is one of the practical reasons why so much depends upon a knowledge of diagnosis; that is, an exact recognition of the factors which bring about chronic obstruction of the common bile duct by concrements and by neoplasms of the biliary passages themselves, of the duodenum, or of the head of the pancreas, etc., enables us to differentiate them.

The very complex clinical picture occasionally makes the decisive differentiation of malignant neoplasms of the liver itself from those of the gall-bladder and of the biliary passages very difficult, as primary cancer of the gall-bladder proliferating into the liver there reaches its greatest development, and as all carcinomata which occlude the duodenal end of the common bile duct, no matter what their origin, give rise to metastases in the liver, etc. The simultaneous description of these various neoplasms, therefore, appears to me to clear the situation but little, and after full consideration of the varying practical indications, I believe a separate description of this subject to be necessary. On account of the prevailing opinions concerning carcinoma of the liver as a primary or secondary

affection, its interesting histogenetic development, its manifold varieties, and probably also because of considerations to which the system of tumors of the liver gives rise, purely clinical interest in the latter is far less; hence the following order of arrangement will be different.

As indicated, we shall discuss manifold clinical pictures, but the combination of the following guiding symptoms will always be observed: General digestive disturbances, and, in connection with these, rapid and unarrested progressive cachexia, the appearance or the absence of a tumor, metastases in the liver itself or elsewhere, cirrhosis of the liver (secondary), compression of the biliary passages, particularly that form accompanied by permanent jaundice, portal vein stasis.

1. NEOPLASMS OF THE GALL-BLADDER

As a type of tumor of the gall-bladder *carcinoma* may be considered, for the other forms of tumor mentioned in literature are either so rare or of such subordinate importance as to be of only slight interest to the physician. *Sarcoma* has the same significance as carcinoma.

Among the granulation tumors of the gall-bladder, *tubercle* must be mentioned (Lanceraux, Heddaeus, Mermann); of benign tumors there are to be considered *true cystic tumors* (Wiedemann), *mucus polypi, submucous fibromata* (Albers), *papillomata* (Riedel, Kümmel), *lipomatous* structures (subperitoneal upon the gall-bladder, Cornil-Ranvier), *aneurysm* of the cystic artery opening into the gall-bladder (Chiari), *echinococcus*; finally, of other malignant growths we must enumerate papillary *myxomata* (Sehüppel), *sarcomata* (Czerny, Riedel, Destrée, etc.).

Cancer of the gall-bladder is a pathologic condition which has only recently been accurately studied. Early descriptions were given by Stoll, Hall, Baillie, Heyfelder and others (1777–1839). The first positive cases were probably described by Durand-Fardel (1837–1840). Villard (1871), in his "Étude sur le cancer primitif des voies biliaires," collected 24 cases of primary cancer of the biliary ducts, of which 21 were carcinomatous degeneration of the gall-bladder. In a subsequent series of reports, the most important are those of J. Krauss and H. Zenker, the latter author having most thoroughly and convincingly described as the most important factor in the etiology of cancer of the gall-bladder the presence of gall-stones. Noteworthy anatomical indications, in addition to those already mentioned, are contained in a publication by Ohloff. The most comprehensive statistics have been compiled by Courvoisier and Musser. Among recent comprehensive publications from a surgical standpoint, that of A. Heddaeus must be mentioned. At the present time, the pathologic anatomy and the symptomatology of cancer of the gall-bladder are well understood, and only a few questions touching the border-line between internal medicine and surgery call for further explanation.

Cancer of the gall-bladder is only very rarely secondary, that is, proliferating into the gall-bladder from neighboring organs (the pancreas, the duodenum, the stomach, etc.). The earlier view (Baillie), according to which the gall-bladder was said to be most rarely attacked by cancer except when the disease had previously developed in the liver, has been entirely abandoned. There are numerous cases on record in which the gall-bladder was the only organ which showed malignant involvement, the liver being entirely free. And this is favored by the fact that cancer of the liver which occurs simultaneously has, as a rule, proliferated by contiguity, or is to be looked upon as metastasis of the gall-bladder. In old complicated cases with disease extending to all the organs in the region of the liver, at the present time the gall-bladder is always assumed to be the primary seat if it show but a moderately severe implication. In such cases we occasionally find in the liver a tumor mass with indefinite, limited borders, embedded in cicatricial adhesions, in the center of which upon incision a cavity is disclosed with irregular borders and a pappy or purulent mass containing concrements. The carcinoma proliferating from the gall-bladder to the adjacent liver naturally gives rise to a tumor which, at first sight, may resemble a primary massive cancer of the liver. Only in one case, Tyson's, was the carcinoma of the gall-bladder of purely metastatic origin, and this growth was found at the autopsy in a case in which three operations for cancer of the lip had been performed.

As a rule, cancer of the gall-bladder arises primarily either from the fundus or the neck, rarely from the middle portion, or the wall adjacent to the liver. It occurs either as scirrhus or adenocarcinoma. The soft cancerous alveoli readily break down and form a malignant ulcer. Colloid cancer (Villard, Aezel, Biach, Roberts, Janeway, Brodowski) and villous cancer (Heschl, Klob, Moxon, Voss, Fifield) occur. Conspicuous, yet particularly mentioned only by Mulat, is the pavement cell epithelium with the well known onion-like microscopic configuration. Either the cancer surrounds the gall-bladder, and only implicates a small portion of the walls, or the entire wall is uniformly infiltrated. Often there is but a small tumor, which nevertheless has become the focus for a large, secondary cancer of the liver, or has produced extensive metastases. The entire malignant gall-bladder has contracted upon one or a few gall-stones. In other cases the gall-bladder appears as a nodular tumor of the size of a fist. Occasionally at its fundus it forms a diverticulum which is separated from the rest of the organ by a thickened ring and contains gall-stones; this ring is usually the seat of the cancerous development. As is obvious from the foregoing, the carcinoma usually proliferates to the surrounding areas. Widely extended dendritic infiltration of Glisson's capsule is not rare. In particular, the adjacent liver, the parietal peritoneum, the omentum and the intestine (above all, the colon) are primarily implicated by pericystitic adhesions, and the secondary growth penetrates the connective

tissue membranes. Extending by way of the hepato-duodenal ligaments the cancer reaches the portal lymph nodes, which become markedly enlarged, and by pressure upon the portal vein produce ascites. Thrombosis of the trunk or of the larger branches of the portal vein is not rare. By continuity through the cystic duct the neoplasm attacks the common gall-duct and the hepatic duct, and by stenosis causes obstruction to the outflow of bile, dilatation of the gall-bladder, and stasis jaundice. The hepatic duct and common gall-duct may also be implicated externally by the carcinoma. In some cases a further distribution into the small intra-hepatic gall-ducts has been observed, so that upon transverse section they appear as cancerous rings. This gives rise to the formation of new bile passages and atrophy in the trabeculæ of the cells of the liver acini, causing the retention of bile in the liver and the formation of small cysts which are covered with the epithelium of the biliary passages. In the further course of the affection, inflammatory products collect. The malignant mass breaks down upon the inner surface, the tissue threads reach the bile, which also decomposes, and is changed into a dirty, grayish-brown fluid admixed with blood and pus. The process of destruction usually extends even further, to the adjacent tumor masses between the stomach, the large intestine and the liver, and thus an ichorous cavity develops which is filled with a dirty, gray mass, permeated with cancerous nodules. Continued stasis of bile and consequent infection produce a distributed cholangitis. Not rarely fistulas form which perforate into the liver, into the gastrointestinal canal, and into the abdominal wall (navel).

Although cancer of the gall-bladder by continuous proliferation is prone to attack the adjacent liver and the biliary passages, as well as quite commonly the porta hepatis, it may also give rise to *metastases,* as I must here reiterate in opposition to the opinion which has been generally maintained since the time of Courvoisier. The metastases are not only found in the liver, but, considering the brief duration of the disease, often in other and quite distant organs (in the peritoneum, the omentum, the regions of the umbilicus and the urinary bladder, the recto-vesical fold, the kidney, the spleen, the intestine, the lung, the pleura, the ovaries, the portal, retroperitoneal, mesenteric, and omental lymph-glands). I know of a case in which an incorrect diagnosis was made because the first and chief objective symptom consisted of tumors which were found in the pelvis.

ETIOLOGY AND PATHOGENESIS

The *etiology* and *pathogenesis* of carcinoma of the gall-bladder therefore necessitate the most minute investigation on the part of the practitioner, because of the difficulties which up to the present time have successfully resisted a surgical treatment of the affection, and which depend, in no small degree, upon the fact that the chief cause of cancer of the

gall-bladder is not sufficiently considered, and operation is generally deferred until too late. Almost all investigators have been struck by the extraordinary frequency, more than mere coincidence, with which stones (or ulcers and cicatrices due to stone) as well as cancer are found in the gall-bladder. Naturally the question arises, What causal relation exists between the presence of gall-stones and the formation of cancer? While some authors were formerly inclined to regard the development of cancer as primary, for 30 years past there has scarcely been a pathologist who doubted that cancer is due to the irritation of stones that have previously formed (Klob, Frerichs, Klebs, Willigk, Schüppel, Krauss, Zenker, Courvoisier, Naunyn, and others). This connection is, in the first place, borne out by statistics. Courvoisier found gall-stones simultaneously with cancer of the gall-bladder in about 90 per cent. of all cases, Siegert in almost all, and Löbker in all cases. According to Schröder, 14 per cent. of all patients with gall-stones are attacked with carcinoma of the gall-bladder; according to Naunyn among 150 such cases, 14 developed cancer of the biliary passages. Of 172 patients with gall-stones operated upon by Löbker, 17 suffered from carcinoma of the gall-bladder, and Riedel assumes that about 10 per cent. of all patients suffering from cholelithiasis subsequently acquire carcinoma. The presence of stones prior to the development of cancer can be proven clinically as well as pathologico-anatomically. It has long been known that carcinomata are prone to form in cicatrices, particularly in the stomach, as has recently been clearly proven by G. Hauser, and H. Zenker conceived the idea that the development of cancer of the gall-bladder might be similarly connected with preceding ulcerative processes which might be attributed to pressure necrosis due to stones. Zenker demonstrated clearly that the successive cicatrices cause an atypical glandular proliferation which predisposes to carcinoma. The increased growth of the remaining glandular portion, the shooting forth of sprouts and their extension in various directions, the more marked lobular formation, are primarily but the expression of a regenerative endeavor of the tissues; the character of the excessive and atypical growths gains prominence by the chronic irritation of the stones. Hauser also considers the absence of physiologic resistance, and Ribbert the constriction of portions of the glands by connective tissue, as important. Ohlof's conception is somewhat different; he believes the metaplasia of the cylindrical and plate epithelium as the result of stone formation is causative. The composition of the newly formed plate cells becomes horny, concentric layers form, and, finally, nest formation takes place. This, however, is probably true in only a few of the cases.

Cancer of the gall-bladder is by no means so rare as was formerly believed; at all events, primary cancer of the gall-bladder is much more frequent than carcinoma of the biliary passages. Approximately, cancer is found in females 5 to 7 times more frequently than in males. Whether,

besides cholelithiasis, the clothing worn by women (lacing) exerts a predisposing influence cannot be determined. Carcinoma of the gall-bladder usually occurs between the 40th and 70th (or possibly the 80th) years of life. In the majority of cases it develops after the age of 50. Frerichs mentions a case of cancer of the gall-bladder in a patient aged 26.

SYMPTOMS

The symptoms, unfortunately, are, in the main, very vague, and the clinical picture of carcinoma of the gall-bladder is by no means typical.

In a certain number of cases, at most in $\frac{1}{5}$, we will learn from the history that cholelithiasis has preceded. But the frequent absence of this proof, naturally, does not disprove the theory that cancer of the gall-bladder has developed from the irritation of stones. As is well known, concrements in the gall-bladder do not always cause subjective and objective diagnostic symptoms, and cases of this kind, particularly, show a tendency to the development of carcinoma. Among the subjective symptoms in 60 per cent. of the cases, pain in the gastric and hepatic regions (gall-bladder) is mentioned, partly paroxysmal, partly of a spasmodic character ("gastric pain"), partly continuous and then less severe. The hepatic region, as a rule, is markedly susceptible to pressure. There is loss of appetite, frequently absence of free hydrochloric acid in the gastric contents, usually constipation, rarely diarrhea. Jaundice of varying degree occurs in about $\frac{2}{3}$ of all cases. Enlargement of the liver develops in consequence of stasis of bile or metastasis. Enlargement of the spleen, particularly in long existing jaundice, is not very rare. Ascites is comparatively frequent; its varying appearance, the jaundice, and the successive parallelism and divergence of biliary and portal vein stasis, I shall consider later. Jaundice is observed more than twice as frequently as ascites. Moderate or even decided fever, invariable emaciation, muscular weakness, cachectic appearance, insomnia, and, with decided jaundice, the hemorrhagic diathesis as well as comatose conditions, are often observed.

Following Courvoisier, the symptoms are designated as "*uncertain ones*"; they might also be called "*inconstant ones.*" In contrast to these, alone or combined with them, tumor of the gall-bladder is a significant sign which makes a diagnosis possible.

Of all the *methods of examination* employed in such cases, palpation is the most important. Percussion usually gives us no reliable data. Relative dulness, at least, can generally be determined over the tumor of the gall-bladder, and, passing upward, this is usually merged in that of the liver. If, however, the intestine covers the elongated, sausage-like gall-bladder between the fundus and the border of the liver, which sometimes occurs, even this relative dulness is absent, or, at least, its connection with liver dulness is uncertain. For palpation, the patient assumes the dorsal

decubitus, draws the knees up, and opens the mouth. The head of the patient should be low, the respiration quiet. The seat of tenderness is slowly palpated, and we become convinced that the median region above the navel and the region of the typhlon (cecum) is not conspicuously sensitive. To prevent muscular contractions, we must gently probe with the tips of the fingers into the deeper portions, the fingers being kept extended. Rheinstein has recently called attention to a method of palpating the gall-bladder. The best results are obtained by bimanual examination with the patient in the recumbent posture, and, if necessary, also while the patient is in an erect position. The physician, standing to the right of the patient, places his left hand upon the right lumbar region. The index finger rests upon the twelfth rib, the tips of the fingers being directed toward the vertebral column. The right hand is placed upon the anterior abdominal wall of the patient, the ulnar border being about parallel with the linea alba. The tips of the fingers palpate toward the lower border of the ninth rib, which brings the second finger to a point corresponding with the parasternal line. With the left hand strong pressure is made upon the lumbar region internally and anteriorly, the right hand exerting pressure at the same time so that the liver is simultaneously forced somewhat downward, and the gall-bladder presses upon the lower half (also presses inwardly) of the right kidney, where it may be palpated. If the patient is standing, the right hand of the physician passes below the anterior border of the liver, so that the four fingers press the fundus of the gall-bladder downward, while the thumb exerts pressure anteriorly and the left hand grasps and helps to immobilize the liver. The latter method is particularly satisfactory if the gall-bladder be but little or not at all enlarged. In Wijnhoff's method of examining the liver, the patient sits upon a chair, flexes the legs, and bends the upper portion of the body anteriorly. The physician is seated at the right of the patient, and palpates the anterior region of the liver, grasping it from behind. Palpation of the gall-bladder is, of course, greatly facilitated by anesthesia. According to Naunyn, if we occasionally fill the stomach with gas, we may be convinced that the tumor in question corresponds to the gall-bladder; in this case it moves decidedly to the right, sometimes also upward; it is also forced more in the direction of the abdominal walls, may be more readily palpated, and even becomes visible. A renal tumor, on the other hand, disappears when the stomach and intestine are filled with air (water). Exploratory puncture, of which Harley, in particular, made improper use, is no longer employed.

In about one-fourth of all the cases, even in the later course of the disease, the carcinomatous gall-bladder cannot be palpated. It is then either covered by the enlarged liver, or has become adherent to the stomach and small intestine, deep in the abdominal cavity, or is markedly contracted. The tumor present is not always actually situated in the gall-bladder

region; its site varies decidedly (hypochondrium and also epigastrium, right mesogastrium, and rarely it is low down at the level of the crest of the ilium). Taylor believed that the enlarged gall-bladder was always situated close to a line drawn from the normal position of the fundus of the gall-bladder (from the anterior end of the cartilage of the tenth rib) obliquely to the left and downward so that the median line is crossed somewhat below the navel. This "diagnostic" line is of no value. The typical form of a tumor of the gall-bladder is oval, but it may also be cylindrical, and a globular, a sausage, or cucumber form is also observed. The upper border of the tumor is usually difficult to define; sometimes, it is true, we may perceive distinctly that the tumor protrudes below the border of the liver; that is, we may trace it to the right and to the left sharply up to the tumor, and also inward from the periphery. The tumor follows the expiratory excursion, and may be displaced laterally; where there is connective tissue fixation, the mobility is lost. Sometimes only dilatation of the gall-bladder is apparently present, although a carcinoma of the wall (neck of the gall-bladder) may exist. The determination of an uneven, nodular, tough composition of the tumor is significant. Fluctuation, particularly with a "smooth" tumor, is frequently found.

DIAGNOSIS, DURATION, AND PROGNOSIS

The *duration* of the disease, from the onset of positive symptoms until death, is scarcely more than from 6 to 8 months, and in about one-half of all cases is less than 4 months.

It is often difficult or quite impossible to recognize carcinoma of the gall-bladder, even in advanced stages of the disease; but, in the majority of cases, by repeated careful examinations, and a minute weighing of all the conditions present, it may be correctly diagnosticated. The tumor of the gall-bladder is occasionally looked upon as a wandering kidney, a tumor of the omentum, as a corset lobe of the liver, and even as cancer of the pylorus. Such errors are caused by the deviating position, the unusual form, consistency, etc., of the tumor. Of much greater practical importance is the fact that comparatively often operation is performed for a supposed cholelithiasis, and laparotomy discloses a more or less well-advanced cancer. In a differentio-diagnostic respect it is here of the utmost importance that, as well as a hard tumor, we may find no sharply circumscribed tumor of the gall-bladder but only a more diffused enlargement (resistance) of the entire gall-bladder region including the adjacent liver, and thus the growth of the tumor can be recognized. Inversely, sometimes enlargement of the gall-bladder from other causes (the presence of stones or a mere thickening of the walls of the gall-bladder) has been mistaken for a malignant growth. In cases in which a tumor in this region cannot be determined with certainty, the liver may be definitely shown to have undergone carcinomatous

degeneration; if no primary carcinomatous disease can be found in other regions of the body, and there is decided pain upon pressure in the region of the gall-bladder, and the patient is a woman, carcinoma of the gall-bladder should be thought of as the underlying affection. If ascites be a complication, splenic enlargement is an important diagnostic factor; if cirrhosis can be excluded, ascites and enlargement of the spleen favor a neoplasm upon the concave surface of the liver. Often the course is calculated to facilitate the recognition of the affection. Examination of the urine and of metabolism does not enable us to form an early conclusion as to the presence of cancer. In carcinoma of the gall-bladder, however, a degree of cachexia soon appears such as is observed in cholelithiasis only late or after permanent occlusion of the common gall-duct. Incarceration from stone may occasionally last for years; a case of carcinoma of the gall-bladder usually terminates in less than six months. If, however, we wait for the time when a hard, oval, nodular tumor appears in an area corresponding to the gall-bladder, and follows the respiratory excursions, and swelling, a hardening, and a nodular consistence of the liver as well as jaundice and ascites have been superadded, the autopsy will then rarely reveal an error in diagnosis, but the time for professional aid will always (or almost always) have passed!

After these statements, the fact that the *prognosis* of carcinoma of the gall-bladder is always exceedingly grave needs no explanation.

TREATMENT

The only *radical treatment* is extirpation of the gall-bladder, and, as a rule, simultaneously more or less extensive resection of the liver. Cholecystectomy was first performed by Langenbuch in 1880, and since that time there have been numerous reports from surgeons who have studied the indications for this operation. The controversies concerning this point do not belong here; but they express the unanimous opinion that extirpation of the gall-bladder for carcinoma, with or without simultaneous resection of the liver, up to the present time has been absolutely unsuccessful. In so far as reports are at hand, almost all the cases operated upon succumbed to early relapse, to metastases, or other complications (Tait, Czerny, Riedel, Bardenheuer, Hochenegg, v. Winiwarter, Socin, Heidenhain, v. Mikuliez, Löbker, and others). Kehr is, therefore, of the opinion that if cancer of the gall-bladder has been determined with certainty (exploratory incision), it is wise to refrain from any attempt at a radical cure. Cystostomy may, under such circumstances, bring about transitory amelioration of the condition; with drainage of the gall-bladder the pain ceases; but, on the other hand, in consequence of the cancer attacking the cystic duct, a distressing mucous fistula develops. Löbker attempted in 4 cases to relieve the pain caused by a simultaneously present empyema of the car-

cinomatous gall-bladder by performing eholecystostomy with a possible removal of the stones. All of these patients died soon afterward, two of them directly in consequence of the operation, and sooner than would otherwise have been the case.

If, therefore, we wait for the time when the diagnosis can be positively made, a successful radical therapy is impossible, because the disease is no longer confined to the gall-bladder alone, or to its immediate vicinity, but has extended to neighboring organs, where it has led to the formation of metastases, to perforation, etc. Heddaeus, therefore, proposes that we regard the symptoms designated by Courvoisier as "uncertain ones" as indications for exploratory laparotomy, and that in the given case we make an exploratory incision into the gall-bladder to determine the condition of the mucous membrane. Thus he would attack the evil at its root, would arrest the development of new formations, and thus avoid the danger of too extensive an operation, which inevitably and rapidly leads to death. As in many cases, we refer the onset of the carcinoma to the fundus, he hopes in this way, even by a partial resection of the gall-bladder, to arrest the further advance of the carcinoma. In all cases in which the neoplasm originates from the cystic duct or from the neck of the gall-bladder, this later method would, *a priori,* be excluded, but the resection of an implicated portion of the liver would not cause insurmountable difficulty. The question whether it is justifiable in cholelithiasis, and under certain circumstances, to undertake an exploratory incision may be answered by every physician in the affirmative, as the danger of this operation is in no sense commensurate with the ultimate advantage gained or with the damage which might ensue from neglecting it. Exploratory laparotomy is justifiable when we have long-continued and severe pain, when the patient is unable to follow his occupation, when there is loss of strength and decided emaciation, when internal treatment is futile, when there are negative or entirely obscure findings in the liver and gall-bladder, and those symptoms are excluded which make occlusion of a common gall-duct by a tumor very likely. Patient and physician will be much less willing in the frequent cases in which carcinoma has led to the development of a determinable tumor with but slight subjective difficulties. I doubt, therefore, that the proposition of Heddaeus will largely reduce the number of persons succumbing to cancer of the gall-bladder.

The difficulties which attend a late operation for carcinoma of the gall-bladder may be met by considering all of the practical consequences of the previously described etiology of cancer, and by directing the treatment chiefly to the removal of the cholelithiasis. No practitioner, at the present time, can escape the conviction that in numerous types of cholelithiasis in regard to which physicians and surgeons, fortunately, have recently more nearly approached unanimity of opinion, the surgeons are the ones most likely to afford relief, and are, therefore, the proper therapeutists. The

operative method which is accompanied by comparatively slight immediate danger to the patient, which demands the sacrifice of an organ not absolutely necessary to life, and which is by far the most important seat of gall-stone disease, which removes the first germs of carcinomatous formation in the wall of the bladder irritated by the stone, is *cholecystectomy*. Performed at the proper time, not only for the threatening carcinoma but also for general *curative* and prophylactic purposes, an extension of the indications for extirpating the gall-bladder in the cases in which cholelithiasis is limited to this organ is desirable. At all events, even chronic inflammatory conditions of the gall-bladder with a thickened wall and those anatomical changes of the epithelium which may be looked upon as precursors of carcinoma, should be included in these indications. Naturally these points must be left for the surgeons to decide; it seems to me that in this respect their opinions are at present undergoing a change. Two cases in which Löbker undertook to perform cholecystectomy on account of supposed empyema, while tissue suspiciously resembling tumor was unnoted by the operator, and in which Grawitz showed microscopically a beginning carcinoma, should increase the number of advocates of early cholecystectomy more than it has. Whether we shall in this way succeed in diminishing the percentage of patients with gall-stone disease who acquire carcinoma of the gall-bladder, future experience will alone decide. One point must certainly be touched upon. It has been explained above that in those cases in which stones remain encysted in the gall-bladder for years, there is a tendency to the development of cancer of the gall-bladder producing actual spasmodic paroxysms without inflammatory processes. The law still operative with many surgeons: "Quiescent gall-stones are not subjects for treatment," must therefore be construed with some limitations. Enlarged gall-bladders tensely filled with stones, particularly where there is a hereditary predisposition of the patient to cancer, should be extirpated on principle. The question is simply this: How does the physician observe this except quite incidentally, and how are we to obtain the patient's consent to an operation if we tell him that, in proportion to cholecystitis which is very frequent, cancer is a very rare disease?

2. OBSTRUCTIONS OF THE COMMON GALL-DUCT BY TUMORS

CLINICAL FORMS

In clinical study it is difficult to separate the consideration of *carcinoma of the biliary passages external to the liver* from that of obstructions of the common bile-duct and of the hepatic duct in general.

Occlusion of the ductus choledochus may result primarily from an *anatomical, abnormal, duodenal insertion and twisting* of this passage at its entrance into the intestine, whereby, in exceedingly rare cases, even in

youthful individuals, a valve-like closure and consequent extreme cystic dilatation occurs (Konitzky). This colossal dilatation of the gall-duct may then form a resistant tumor in the right half of the abdomen, and the bulging wall of the passage in itself causes an increasing compression from without upon the end of the gall-duct extending into the intestinal wall. In a child aged 2 months, with decided jaundice, an exploratory laparotomy was performed (by Witzel), and the subsequent autopsy showed a *congenital marked narrowing of the cystic and common ducts, as well as the absence of the hepatic duct. Congenital obliteration* of the ductus choledochus, probably as the result of *fetal syphilis,* is somewhat more frequent. Soon after birth such children are decidedly jaundiced without fever and rapidly perish. This has sometimes repeatedly occurred in the same family (Donop, Binz, Glaister, Schüppel).

Chronic occlusion of the ductus choledochus by stone (*incarceration of calculi*) and the infrequent *cicatricial obliteration* of the same in consequence of cholelithiasis are of far greater practical importance. The very rare *adhesions* due to *propagated* (?) *inflammations* from the intestine in enteric fever (Lebert), in enteritis (Musser), in the puerperium, chronic peritonitis (Andral), and in benign pyloric stenosis (Richard) at least deserve brief mention here. The greatest interest attaches to *neoplasms of the common bile-duct.* We must also mention occlusion by *foreign bodies other than stones,* and by parasites (echinococcus cysts, asearides, and varieties of distoma). *Hydatid cysts* always develop in the liver, from which they penetrate to the larger biliary passages. Years ago a patient of mine was operated upon by v. Eiselsberg for echinococcus of the liver. On account of extreme chronic jaundice which was simultaneously present I diagnosticated a closure of the common gall-duct by daughter-cysts; hepatic echinoeoccus cysts and a cholesterin stone were found in the gall-duct and were at the same time removed. Besides occlusion from stones the hepatic duct is sometimes occluded by *callous cholangitis* (rare), by *neoplasms,* by *fetal syphilis* and by *parasites.*

In addition to the obstructions to the permeability of the large biliary passages which have been mentioned, there are numerous "vinculo quasi injecto" which *compress* the lumen (or simultaneously compress and infiltrate it). These are either actual neoplasms, or tumors in the widest sense of the word, and connective tissue proliferations. These obstructions may originate in the *porta hepatis*: *hydatids, aneurysms* of the hepatic artery (Niewerth), of the abdominal aorta (Stokes), of the superior mesenteric artery (Wilson, Gairdner), enlarged *tuberculous, carcinomatous, lymphomatous, amyloid* glands of the porta hepatis, along the hepatoduodenal ligament, and near the mouth of the common gall-duct, circumscribed adhesive perihepatitis at the hilus in affections of the liver, *syphilis ad basin, ulcers of the stomach and duodenum, mediastinitis* after carcinoma of the breast (Smolenski), and carcinomata. Besides compression,

cicatrices in the region of the porta hepatis also cause torsion of the common gall-duct, so that it is bent and follows an irregular course. We must also consider changes in the size of the *pancreas* (cystic dilatation of the duct of Wirsung (Wyss and others), *suppurative pancreatitis* (Frison), sclerosis (Déjerine), principally, however, *carcinoma of the head of the organ,* and, finally, *malignant disease* and *cicatricial stricture of the duodenum.* Primary *carcinoma of the gall-bladder* may by continuity creep through the cystic duct to the common duct and the hepatic duct, and, besides producing angiocholitis, may cause compression and stenosis. Occasionally the enlarged tensely filled gall-bladder (in consequence of occlusion of the cystic duct by stones), or dropsy (empyema) of the gall-bladder without concrements, may compress the common duct (Ramskill, Socin, Kümmel). Stones embedded in the cystic duct may also occlude the common duct (Wilson, Bardenheuer). Even the relaxed gall-bladder whose tonus is impaired by long-continued distention due to inspissated bile, or by a condition of fulness of the common gall-duct, may cause compression leading to biliary stasis (Petit, Jacob and Cyr). Occasionally such a gall-bladder may be expressed. Adhesions of the gall-bladder to the duodenum and colon may cause a volvulus of the ductus choledochus, and the duodenum, in particular, is drawn to the right and upward. Such *twisted stenoses* are often increased by the simultaneous presence of stones. It is also possible that a *wandering kidney* by its tense ligaments may cause a volvulus of the common bile-duct, a possibility which Weisker denies, but which, nevertheless, exists. On the other hand, tumors of the right kidney may cause obstruction (Schüppel). Even tumors of the colon, the omentum, the retroperitoneum, and of the ovary and uterus, must be considered as possible obstructions to the flow of bile.

If we discuss only the cases in which occlusion of the common gall-duct develops an actual, extensive (absolute), biliary stasis, we find aneurysms and enlargement of the glands of the porta hepatis, as well as cysts of the pancreas, comparatively rare. *Perihepatitic* and *pancreatic indurations* are, as Courvoisier states, more important. Of most serious import in this connection are *concrements and neoplasms in the ductus choledochus, carcinoma of the head of the pancreas and of the duodenum* in the region of the plica Vaterii. *Secondary malignant infiltrations* of the biliary passages by *carcinoma of the gall-bladder* must also be included here. The most significant causes of obstruction of the choledochus are also the most frequent, and, therefore, of the greatest clinical interest. The ductus choledochus in its passage to the duodenum burrows for itself a furrow in the head of the pancreas, or it perforates the same, and unites with the ductus Wirsung near its mouth. The upper horizontal portion of the duodenum, as well as the two other portions, are reached from the head of the pancreas with which the two duodenal areas closely unite and form a bow-shaped intestinal portion. The point where the common gall-duct

enters the duodenum is the left concave surface of the pars descendens duodeni. If the ductus choledochus does not penetrate the head of the pancreas it passes behind it, and enters the substance of the pancreas just before its junction with the pancreatic duct. The two passages run alongside each other for a short distance, then perforate obliquely into the longitudinal musculature, continuing their course for some time below the mucosa, and finally, by a common orifice, pass into the diverticulum of Vater. Sometimes, however, they terminate separately. Compression is most likely to occur when the ductus choledochus passes through the head of the pancreas; but these topical relations explain why a tumor which has developed in the ductus choledochus furnishes a picture so nearly analogous to that of a tumor at the head of the pancreas, or of a carcinoma, or even of a cicatricial stricture in the region of the diverticulum Vateri duodeni. From an anatomical standpoint the question has arisen (Pic) whether carcinoma of the diverticulum of Vater should not be referred to the pancreas rather than to the duodenum. No matter what the conditions are, the obstructions which obliterate or compress the ductus choledochus must also mechanically implicate the pancreatic passage, which is not infrequently subject to dilatation (cystic).

In practice the question usually resolves itself as follows: Jaundice (stasis) reaches its acme of development either rapidly or gradually; if it does not disappear after a short time, it merges into a chronic icterus. In prolonged *"catarrhal" jaundice* (lasting 2, 5 or even 9 months), when the symptoms of gastrointestinal catarrh have subsided and the signs of pure, chronic, biliary stasis have become prominent, particularly a disturbance of the general condition, with weakness and emaciation, the clinical picture of biliary occlusion from other causes (incarceration of stone compressing or occluding neoplasms) may resemble it so closely that the diagnosis is made clear only by the further course of the affection (final, complete and permanent restitution of permeability of the biliary passages). Certain diagnostic support will be found in the fact that the onset of the disease, according to our own observation, or after obtaining a history of the cause and clinical behavior, coincides fully with that of catarrhal jaundice; that the occlusion of the biliary passages is incomplete, having been only partial for a time, or even continuously; and that, finally, the liver and spleen have permanently presented only a sign of biliary stasis. But in catarrhal jaundice the hemorrhagic diathesis may also develop! Neusser and Toelg report the case of a man, aged 39, who was attacked by jaundice after partaking of sausages, and who succumbed in the eighth week of the disease after hemorrhage from the urinary passages. The autopsy showed catarrhal cholangitis, with complete impermeability of the ductus choledochus. The hemorrhages began during the seventh week. Nowadays, under similar circumstances (probably, however, before the appearance of the cholemic hemorrhages) an operation (cholecystotomy)

would be resorted to. As in simple "catarrhal" swelling, a *diphtheritic*, a *typhoid*, and a *variolous* enlargement of the ductus choledochus may remain for a long time. I observed a case of scarlatina with jaundice and decided extension of the gall-bladder. Apart from such cholangitic conditions as produce the syndrome of chronic jaundice, there are certain pathologic processes in the *liver itself* which must be considered in diagnosis: *Echinococcus, abscess, syphilis* (peripylephlebitis syphilitica), *primary and secondary carcinomata*, all at least under the supposition of a corresponding localization, an especial development, or in combination with other (secondary) disease of the tissue (biliary cirrhosis), also *hypertrophic hepatic sclerosis* (Todd-Hanot) in which latter disease the jaundice at first recurs, later becomes permanent, and varies periodically in intensity. Particularly in *hypertrophic hepatic cirrhosis*, which among the previously mentioned processes with by far the greatest frequency runs its course with most decided chronic jaundice, the discoloration of the skin is rarely as green as in complete biliary stasis, and, except during the time of the so-called *poussées*, a sufficient amount of bile enters the intestine to color the feces. A comprehensive description of the differential diagnosis of all the hepatic affections which have been mentioned (besides Hanot's cirrhosis, *primary cancer of the liver* and *echinococcus multilocularis* require special consideration) cannot be given in the framework of this article. All these, however, must first be excluded before we can definitely conclude in a given case that the chronic icterus is due to a definite (quasi definite) occlusion of the extra-hepatic ducts, and observation of the further course will determine which of the many diseases named is the cause. It must, nevertheless, be casually mentioned that, especially to these primary affections of the liver, extra-hepatic complications are usually added which may in themselves cause chronic jaundice (inflammatory swelling; suppuration of periportal lymph nodes in liver abscess, the affection having previously run its course without jaundice; circumscribed adhesive perihepatitis at the porta hepatis in gummatous syphilis of the liver; carcinomatous infiltration of the periportal glands in cancer of the liver or gall-bladder).

Much more important than the exclusion of primary disease of the liver is the diagnosis of the situation of an extra-hepatic constriction of the common bile-duct in the special cases in which jaundice develops with more or less rapidity and intensity, and culminates in the serious condition known as icterus gravis. The color of the skin, even when the jaundice varies or even occasionally becomes intermittent, is greenish, olive color (melasicterus), of the urine dark brown (greenish-brown), of the stools absolutely acholic. In other cases, particularly in the later stages, the discoloration of the skin is less intense, revealing a peculiar tint (a dirty, yellowish- or brownish-red), the urine also appears conspicuously changed to a brownish-red (*ictère hemaphéique* of Gubler, metapigmented icterus

of Debove and Achard, previously called by us "urobilin icterus"). What pigments produce the staining in these cases is not known with absolute certainty. The French declare the principal coloring agent of metaicterus to be urobilin and its chromogens. The former they believe to be exclusively produced in the liver, and its appearance to denote a serious affection of the liver cells. In specially prolonged cases the ieteroid discoloration later loses its intensity simply by loss of function of the liver cells and the consequent insufficient production of bile. The general nutrition suffers to the highest degree because of various digestive disturbances and marked cardiac asthenia; not rarely there is a tendency to capillary, but often very decided, even fatal, hemorrhages, or to those which may cause a fatal termination, such as hemorrhage into the stomach, the intestine, the abdominal cavity, the retroperitoneal cellular tissue, from the urinary passages, the uterus, the nose, the gums, the skin, and from operative wounds (Leyden, Feltz and Ritter). Toward the end there are frequently paroxysms of disturbances which usually terminate fatally in a short time (dizziness, coma, or a state of excitement, delirium or convulsions, and which are at present designated hepatic autointoxication or hepatargy [Quincke]). Rupture of the biliary passages which are dilated to an extreme in consequence of the absolute closure of the common bile-duct is exceedingly rare, but more frequent if, at the same time, suppuration of the biliary passages or abscess of the liver, or, exceptionally, trauma occurs in connection. Distributed infectious processes in the biliary passages are more frequent in occlusion of the common gall-duct by stone or tumor. Ulcerative perforation of the biliary passages is observed almost exclusively in incarceration from stones. Peculiar to cholelithiasis also is spontaneous recovery by the formation of a fistula (between the biliary passages and the intestine, especially the duodenum and colon, or between the gall-bladder and abdominal walls). By direct chemical action of the engorged bile upon the liver substance, biliary cirrhosis ensues (which must not be confounded with cirrhosis cholangica [hypertrophica] Hanot). Marked tissue proliferation and consequent induration, with occlusion of the common duct by stones, occurs more often than in occlusion of the ductus choledochus by carcinoma or cicatricial stricture of the duodenum in the region of the diverticulum of Vater. With biliary stasis due to incarceration of calculi, an infection of the biliary system is attributed to intestinal bacteria which appears to be substantiated by the frequent complication of marked purulent inflammation, and this circumstance chiefly causes, that is to say, favors, the interstitial proliferation of connective tissue. Evidently, however, the French designation of cirrhose biliaire as "calculaire" is much too narrow. If an obstruction by stone leads to true granulation, even to contraction of the liver, to enlargement of the spleen and ascites, the condition is due, in addition to the causes that have been enumerated, to the prolonged course of the disease (sometimes two to three

years). As a rule, this cirrhosis is without decisive importance for the *decursus morbi.*

If, however, in consequence of any occlusion of the common bile-duct, marked, high-graded icterus stasis develops, there are also quite isolated, paradoxical cases in which this condition does not follow. Such a case, for example, was observed by Hanot and Gombault. In consequence of "chronic gastritis with submucous sclerosis and callous retroperitonitis," cirrhosis of the hilus of the liver had formed and completely obstructed the ductus choledochus, but there was no jaundice, and merely partial occlusion of the hepatic artery and portal vein. Hanot and Gombault assumed that the succeeding disturbance of circulation had limited the quantity of bile formation and, synchronous with the hindrance by occlusion, absorption was suspended so long as even the smallest quantity of bile passed into the intestine; later the secretion ceased entirely, so that even complete occlusion of the common bile-duct did not produce jaundice. From a deficiency in formation products, the secretion of bile ceased, and this caused the death of the patient. In this case, the important factor was the simultaneous strangulation of the hepatic vessels. However, I believe it more likely in such exceptional cases that an auxiliary system of biliary canals, which is also present under normal conditions and serves the purposes of anastomosis, intervenes and permits the vicarious emptying of an auxiliary branch of the ductus choledochus and the like.

Among the previously mentioned pathological processes which with comparative frequency cause obstruction of the choledochus the following must be mentioned: *Chronic impaction by stone, carcinomata of the common bile-duct, of the head of the pancreas and duodenum, as well as cicatricial stricture in the region of the diverticulum of Vater, circumscribed perihepatitis at the hilus of the liver, and, possibly, also enlargement of the periportal lymph nodes.* In so far as a gastric cancer, or a primary carcinoma of the gall-bladder, attacks the ductus choledochus and the hepatic duct, the resulting stasis of bile must be ascribed to the tumor of the choledochus provided a fundamental diagnosis cannot be made. It is of the utmost importance for the physician to know with certainty that an occlusion of the biliary passages by stone or some other cause (tumor) has occurred. If we feel assured that a stone is present in the common duct, and in case intermittent fever sets in soon after the first colic which is accompanied by jaundice, choledochotomy is to be performed. In chronic obstructive jaundice due to carcinoma, much less is to be hoped for from operative interference; even exploratory laparotomy, according to experience, is badly borne! Causes purely mechanical also favor the conception of these two groups of cases: *Calcareous occlusion and occlusion of the biliary passages from other causes.* For, as will be shown in the following, a series of other factors may be recognized by which, aside from numerous, not rarely insurmountable, difficulties in

individual observations, the differentiation between chronic occlusion of the ductus choledochus by calculus and tumor may be indicated; as a rule it is almost impossible to distinguish the different cases of occlusion not due to stones. The course is most favorable, as was first indicated by Courvoisier, Leube, and others, and corroborated by Neusser in the diagnosis of *obstruction of the choledochus from pressure in the porta hepatis.* Probably in most of the cases belonging to this group, but by no means exclusively, there is a determinable, characteristic factor, that is, the parallelism between biliary stasis and portal vein stasis evidently caused by the anatomical situation referred to. As symptomatic expressions of the latter, besides ascites and enlargement of the spleen, we must consider diarrhea, gastric and intestinal hemorrhages, and a visible collateral circulation. An intense and almost simultaneous stasis in the portal vein and biliary system (after excluding ordinary hepatic cirrhosis) would lead us to think of enlarged glands at the hilus of the liver and circumscribed perihepatitis *ad basin.* Apart from carcinoma and syphilis, the clinical interest of these processes is comparatively slight. Naturally there are tumors also upon the under surface of the liver, as, for instance, in the case of Quincke, in which a large fibroma was present, but which, on account of its position, merely occluded the choledochus without causing ascites. On the other hand, in the much more frequent cases in which the greatest pressure is on the duodenum, this is a variable condition. Usually jaundice is present for a long time before ascites appears, or the latter symptom does not occur at all (it is most readily produced in carcinoma of the ductus choledochus and of the duodenum itself), that is, during the entire course of the disease it is very slight. Whether the peritoneal effusions of the tumors which we have an opportunity of observing, and which are due to the very small tumors of the intestinal portion of the biliary passages, actually depend upon purely mechanical obstruction of the circulation in the portal vein, I believe to be very questionable. In carcinoma of the head of the pancreas and the upper end of the duodenum, as well as in cancer of the gall-bladder which extends in the direction of the porta hepatis, at least occasionally and in certain stages of the course, the ductus choledochus and the portal vein sustain almost equal pressure.

As is shown by the statistics of surgeons up to the most recent times, in tumors which occlude the distal portion of the choledochus, a positive, diagnostic recognition in the individual case prior to the operation (that is, during the life of the patient) was frequently impossible; if, indeed, a correct opinion with a special localization of the tumor could be given at any time. On the contrary, the operation was usually performed because, diagnostically, the disease was supposed to be present, but, at the same time it was often erroneously presumed to be a case of occlusion of the biliary passages by stones. I shall, therefore, utilize a somewhat extensive and original experience in regard to obstruction of the chole-

dochus by tumor to criticize the differential diagnostic factors which have up to the present time been proposed. Since 1887 I have collected 24 autopsy reports of occlusion of the choledochus not due to stone (in Prague, Vienna, Graz). In another case, metastases due to sarcoma were present, which, starting from a large, visible and palpable tumor of the left ilium, compressed the biliary passage. Twenty-two cases were due to obstruction from carcinoma, the two other cases were attributed respectively to an echinococcus cyst upon the concave surface of the liver and to a cicatricial stenosis of the duodenum after rupture from a stone. In 13 cases we were dealing with cancer of the biliary passages, in 7 with carcinoma of the head of the pancreas, and in 2 with a duodenal tumor. To this must be added 2 cases (with section) of tumor of the pancreas without jaundice, and 3 others, not confirmed by autopsy, of occlusion of the ductus choledochus, in which the diagnosis of carcinoma was made with at least great probability and which, in certain respects, may here be utilized. In contrast with these reports of occlusion of the choledochus not due to stone are 11 original cases in which, by autopsy or operation, incarceration due to stone, to cicatricial stricture of the biliary passages following cholelithiasis or abscess, but without a complicating carcinoma, could be determined. From the literature at hand it is difficult to form a conclusive opinion as to the frequency of chronic occlusion by stone compared with that due to pressure from malignant tumors. Hilton Fagge, after an experience extending over years, states that permanent biliary stasis with all of its sequelæ occurs twice as often in consequence of malignant disease causing pressure as in pressure from stones. Murchison knew of only two cases of fatal, uncomplicated, chronic jaundice due to stone, and Glover believes every case of this kind worth publishing on account of its great rarity. Robson cautions us in general against operations for obstructive jaundice, since, as a rule, carcinoma of the biliary passages is found. Fiedler found this condition only twice among 800 to 900 autopsies in cases of gall-stone disease (that is, among 10,000 general mortalities). In 343 cases of cholelithiasis (among 4,313 autopsies) Schloth (in Erlangen) found a stone in the ductus choledochus 9 times, therefore, in 0.2 per cent. of all autopsies, and in 2.6 per cent. of all the cases of gall-stones. According to Courvoisier, the proportions are even greater in Basle: In 2,520 autopsies and 255 cases of cholelithiasis, stone was found 10 times in the choledochus, i. e., in 0.4 per cent. of all autopsies and 3.9 per cent. of all gall-stone cases. The rarity of pure cases coming to autopsy does not certainly prove the rarity of the condition; occlusion due to stone need not, as such, be absolutely fatal, for, in a large number of cases, the calculus eventually finds its way out. Of 6 non-operative cases collected by W. Braun (Pringle, White, Copeman, Michaux, Riedel, Thornton) the outcome was fatal in 5, and in the patient of the last named author a spontaneous passage of the stone occurred and

was followed by cure. Important complications may set in. That chronic jaundice due to a calculus permanently incarcerated in the ductus choledochus is not *absolutely* rare is proven, in the first place, by the experiences of surgeons who are specialists in this line. Nevertheless, according to my own experience, if the physician does not observe "selected" cases, even allowing due weight for the intermissions in the jaundice, he must be familiar with the fact that the severest (fatal) cases of chronic biliary stasis are due far more frequently to obstruction of the choledochus which is not due to calculi. In this opinion Schüppel coincides.

PATHOLOGY

In regard to the special pathology of *adhesive perihepatitis in the porta hepatis* and in *affections of the periportal lymph nodes,* I shall not at this point enter into details. In syphilitic hepatitis marked icterus from compression of the biliary passages is much rarer than ascites. Carcinomatous metastases of the portal glands are peculiar to primary and secondary cancer of the liver, and cause decided swelling. Primary cancer of the gall-bladder leads to the development of cancer in the glands of the hilus and in the porta hepatis, by which the outflow of bile is impeded and the portal vein is compressed. Ascites is found here much more frequently than enlargement of the spleen. Intestinal hemorrhage, even dilatation of the collateral venous trunks, is observed in this condition. It must be borne in mind that the portal vein may also be occluded and ascites result from complicating cirrhotic changes (carcinomatous cirrhosis) or from obstruction (compression) of the veins of the liver. In leukemic and lymphomatous tumors, the portal lymph-glands are also frequently enlarged. But if the liver is not correspondingly changed from other causes (for example, in certain cases of pseudo-leukemia with chronic relapsing fever) decided jaundice is very rarely present, and ascites usually occurs late. (Chronic peritonitis of leukemia belongs to another category.) In the diagnosis of these conditions, therefore, aside from the relation between the biliary and portal vein stasis, it is necessary, above all, that we find in some part of the body the primary organic affection which would produce secondary carcinomatous disease of the glands of the hilus, or the proof of an analogous affection of other peripheral lymph-glands (leukemia, pseudo-leukemia), or that we discover signs of constitutional syphilis, etc.

The case mentioned above of *compression of the biliary passages* upon the lower concave surface of the liver by an *echinococcus cyst* may be here briefly quoted.

A woman, aged 30, wife of a meat-chopper (butcher), who had previously suffered from enlargement of the liver and ascites, and afterward from gradually increasing jaundice, subsequently noted a more rapid and more decided enlargement of the abdomen with slight swelling of the feet. Severe abdominal pains were added.

On June 8th, 1888, she was visibly emaciated, markedly jaundiced, her color brownish-gray, her temperature between 100° F. and 102.9° F. Upon the right side of the neck a mass of partly isolated, partly connected, lymph-glands of the size of a small apple; in the left supraclavicular fossa isolated intumescent lymph-glands from the size of a millet seed to the size of a bean. Left-sided pleurisy. Over both lungs piping râles, occasionally crepitation. Free fluid in the peritoneal sac. The liver enlarged, of coarse consistence, its surface smooth; the border, which may be readily palpated, is dull. Gall-bladder not palpable. On percussion, at least, enlargement of the spleen. Urine very dark, yellowish, foamy; chlorids in normal amount.

Pain in the right hypochondrium continues. The temperature rises each evening above 102.2° F., early in the morning it falls below 100.4° F. Feces not entirely devoid of bile, very rich in fat. No bacilli in the expectoration. June 18th: Pericarditis. June 19th: Complete absence of bile from the feces. Hemorrhagic diathesis, hemorrhages into the skin, from the nose, hemorrhage of the gums. June 23rd: Marked loss in strength. June 25th: Subnormal temperature. June 26th: Death.

Autopsy: Echinococcus cysticus multiplex cum compressione ductus hepatici et cystici, subsequente icteru gravi.—Tuberculosis chron. gld. lymphatic. coli. Pericarditis, pleuritis sin. tuberculosa cum exsudato fibrinoso-haemorrhagico. Peritonitis tub. The liver appeared enlarged to 1½ times its normal size; upon the upper border of the right lobe between the gall-bladder and the hepato-umbilical ligament an uneven tumor of the size of a man's fist was noted, and at the vault of the liver, under the capsule, a whitish tumor the size of a hen's egg was found. The first mentioned tumor bulged decidedly downward, so that around it the hepatic duct and the beginning portion of the common duct were found empty and pressed flat. Owing to this dragging, previously mentioned, it was difficult for the sound to pass the biliary passages. The cystic duct also encircled the lower border of the tumor, and this, too, markedly dragged. The walls of the distended biliary passages were decidedly atrophic. The gall-bladder contained but little bile. A third tumor of the same character was found deeply embedded in the right lobe of the liver; the liver tissue showed an increased consistence. A brownish-green icteroid condition was observed. The distended intrahepatic biliary passages contained much mucoid, watery, yellow bile. The enlargement of the spleen was very considerable. The tumor presented the characteristic features of echinococcus cysts.

As is well known, compression of the large biliary passages upon the concave surface of the liver combined with jaundice in consequence of the stasis of bile is rare, particularly in echinococcus cysts of the liver; according to Finsen it occurred only 7 times in 167 cases, according to Neisser 20 times in 388 cases. An analogous effect upon the portal vein or vena cava is infrequent. Damaschino once found a large echinococcus sac in the right lobe of the liver, pressing upon the ductus choledochus in the porta hepatis; in this case another small hydatid lay along the longitudinal fold of the bile ducts. In my case the complete closure, the icterus gravis, the hemorrhagic diathesis, the hepatargy, as well as the preceding ascites (apparently the peritonitis occurred late), were most remarkable.

I have seen no case in which *enlargement of the pancreas other than from carcinoma* could give rise to complete occlusion of the common bile

duct. Evidently obstructions from cystic dilatation of the duct of Wirsung (Wyss) from purulent pancreatitis, and even from sclerosis, are rarities. On the other hand, tumors of the head of the pancreas with actual tumors of the choledochus may in their frequency enter into competition (in this relation cancer is first, lymphosarcoma is rare). Many authors regard cancer of the pancreas as even the usual cause of obstruction of the common bile duct. In my own observations, as has been mentioned, there are 13 cases of cancer of the choledochus (among these 8 positive primary neoplasms), and, in contrast, 7 of primary carcinoma of the head of the pancreas. No doubt primary cancer of the duodenum beginning at the diverticulum of Vater is much more uncommon. One of my patients with cancer of the pancreas was only 25 years old, the age of the others varied between 47 and 70. In regard to sex my experience was uniform with that of all other observers, in that among my cases there were but two women. Of the etiology of carcinoma of the pancreas, we know as little as of the cause of cancer in general. Anatomically, the epithelioma starting from the epithelium of the glands as well as from the cells of the ducts usually presents itself as scirrhus, more rarely as a medullary tumor, and most rarely as a colloid cancer. Most often (in over 60 per cent. of all cases) the seat is in the head, and the tail of the gland is usually exempt. The growing neoplasm of the pancreas much more frequently proliferates to the duodenum (also to the stomach) than the duodenal (pyloric) cancer to the pancreas (in spite of Olivier's conclusions). The head of the pancreas attacked by cancer is often decidedly enlarged, up to the size of a child's fist or larger; during the life of the patient, however, even to the end, the tumor is not palpable (this was true of 6 out of 7 of my cases), which is in contrast to the view of Leichtenstern, who maintained that it was very frequently palpable. Sometimes the pancreatic duct is occluded, and if the resisting tissue still secretes fluid, stasis of the secretion with cyst-like dilatation of the duct occurs (Bolds). As may be readily surmised from the previous description, the ductus choledochus with very rare exceptions meets the same fate, but the mode of implication of the biliary passage varies. If this reaches the intestine without entering the substance of the pancreas itself, under some circumstances compression alone occurs. The common bile duct remaining free may, however, be actually surrounded or infiltrated by the carcinoma. If it permeate the head of the pancreas, a malignant growth develops in its walls, perforates it, and occludes the lumen. Under these varying conditions decided stenosis of different grades is found rather than absolute closures. Dilatation of the biliary passages is the rule, dilatation of the gall-bladder is found in at least 70 per cent. of all cases that have been observed, in all of my own cases with the exception of one (Clässen, Arcelet, Krieger, Legendre, Rotch, Lachmann, Wyss, Frerichs, Murchison). Besides cancer of the pancreas, I saw one case of chole(cysto)lithiasis.

The large blood-vessels in the vicinity and the solar plexus are also implicated in cancer of the pancreas, and not rarely, as is the case with the duodenum; in my 7 cases perforation of its wall was found 3 times. Even the ureters may be compressed; this is partly due to pressure, partly to continued carcinomatous growths, and, in some measure, to the development of tough, strand-like thickenings, with volvulus of portions of the intestine, etc. Apart from the mesenteric lymph-glands and the peritoneum, extensive metastases in distant organs are said to be rare; I found several quite isolated ones and even general carcinosis. In three instances I saw numerous secondary cancerous nodules in the liver. At the autopsy of one of my cases a tough blood clot was found which had been lodged behind the stomach and correspondingly presented the shape of a half-filled stomach. The blood originated from the malignant pancreas, upon the posterior wall of which and adjoining the blood focus, two lacerations were found, more than 3 cm. in length and running a zig-zag course through the serosa and the sub-serous connective tissue.

At least briefly, both observations of *carcinoma of the pancreas* are to be mentioned in which the ductus choledochus *remained free.*

The *first* of these cases occurred in a cheese-maker, aged 59, who was repeatedly the subject of trauma of the abdomen. Upon muscular exertion the patient suffered from pain of a neuralgic character in the upper abdominal region, deeply localized between the pit of the stomach and the navel, and radiating toward the back. For four months he had severe diarrhea, and there was great emaciation (loss of 13 kilograms). Upon the 5th of November, 1898, there was no fever, no jaundice, no palpable abdominal tumor, no ascites. Liver enlarged and permeated with nodules. Gall-bladder not palpable. Spleen of normal size. Except in the inguinal region, no enlarged glands. No edema. Four hemorrhagic stools daily.

By the administration of two test-meals the amount of free HCl in the gastric contents was proven. The stomach was not dilated. The diarrhea was somewhat improved (milk diet). No butter stools; no marked alimentary glycosuria. Up to the time of death (November 19th) a further loss in weight of 5 kilograms.

Autopsy: Carcinoma of the body of the pancreas, the head remaining free. Beginning rupture into the stomach. Hepatic metastases.

The *second* case was that of a locksmith's apprentice, aged 18, who, after quite prolonged constipation, was attacked toward the end of May, 1900, by colic neuralgia and vomiting. The patient observed a rapidly growing tumor in the upper abdominal region; some portions of the feces (after purgatives) were said to be "white like bacon." Upon the 7th of June, the patient was still well nourished and without jaundice. There was no fever, no edema. Bilaterally upon the neck and also in the right axilla small coarse lymph-glands were palpable. A little to the right of the epigastrium, between the median line and the arch of the rib, was a prominence about 8 cm. in diameter, coarse, globular, but nowhere fluctuating, and insensible to pressure. The tumor was deeply adherent, situated in front of the abdominal aorta behind the stomach, readily displaced laterally, and also somewhat movable upon change of position and with inspiratory excursions. Liver and spleen not enlarged. The colon which, on account of inflation with gas, could be readily seen and palpated, surrounded the tumor from below, and in front the stomach appeared to be forced upward. The latter organ was not dilated.

Constipation continues. The stools are stained with bile; they contain numerous unchanged muscular fibers and starch granules, and are apparently rich also in fat. (Fat-splitting not investigated.) The urine contains no indican. The gastric contents (2 test-meals) show absence of free HCl; the total acidity varies between 1.1 to 2.1 to the thousand HCl. As the filtered gastric contents have a brownish appearance, the absence of free acid is probably due only to regurgitated bile. Decided alimentary glycosuria cannot be proven. The tumor grows rapidly. On account of the torturing attacks of neuralgia coeliaca, and at the urgent entreaty of the patient, exploratory laparotomy was performed (Prof. Nicoladoni). This demonstrated that the duodenum had been considerably narrowed by the tumor. Death upon June 28th.

Autopsy: The tumor to the left reaches to the duodeno-jejunal flexure which appears to be forced to the left and upward. The duodenum also appears to be displaced to the right. The permeable ductus choledochus runs its course upon the right surface of the tumor. The right portion of the pancreas is granular, tough, and shows a whitish discoloration; the tumor extends across this portion of the head, and includes a portion of the retained tail of the gland. Two-thirds of the pancreas has been obliterated by the neoplasm, the duct of Wirsung is markedly dilated. In the vena lienalis is a non-obstructing thrombus from the tumor.

I believe it is not too much to declare in these two cases, in the first on account of the typical pains, the diarrhea and the normal gastric function, in the second from the topical relations of the tumor and the fact that biliary stasis did not occur, that a probable diagnosis is possible. Furthermore, in both cases the reason for the absence of obstruction of the choledochus is quite obvious; the carcinoma did not develop from the head of the pancreas. No explicit and unanimous reports are found in literature to show how frequently persistent impermeability of the duct occurs in cancer of the pancreas involving the body of the gland, but the most reliable reports are furnished by Wyss.

Similarly as in the vessels, a carcinoma of the liver may directly implicate the *biliary passages,* forming a tumor mass extending into the large biliary passages and the intrahepatic ducts, and leading to occlusion of the same, thence to biliary stasis. Benign neoplasms arising from the ductus choledochus and the hepatic duct were early mentioned in literature (the reports of Courvoisier are not complete, Alexander Benedictus, 1508, Marcellus Donatus, 1588). Valuable descriptions have been published since 1840 (Durand-Fardel, W. Stokes, van der Bye). Among the forms described were "fibroids" (Albers), "fatty tumors," i. e., presumably lipomatous tumors (Wardell, Bouisson), fibrosarcomata (metastatic, Schüppel), adenomata, myoadenomata (Calzavara), cystoadenomata (Hippel). As in the bile organs in general, here also *carcinomata* are far the most important tumors, and the ones most frequently found. In a number of cases, it was microscopically proven that they originated in the glands of the bile ducts. The large bile ducts, particularly the central portions, are well supplied with glands. These are found in profusion in the hepatic duct, are less plentiful in the upper portion of the ductus chole-

dochus, and in its lower portion least numerous. The body of the gland, constructed of acini, lies like the crown of a stone pine in the connective tissue walls (Riess). A mucoid secretion is ascribed to it, but it is still doubtful whether it contains true mucin. After enlargement of the gland vesicles, perhaps by coalescence, the cylindrical epithelium proliferates markedly, and permeates the lumen in the form of tubes. Connected with this is a proliferation into the stroma, and the gland vesicles are transformed into microscopically small, cancerous nodules (M. Howald). Usually the stroma is profuse, and the cancer shows the characteristics of scirrhus (Dieckmann). In rare cases the conditions are those of colloid cancer. The neoplasm first filtrates the wall of the biliary passage which it narrows and later occludes. The mucous membrane above the infiltrate may for a long time remain wholly or partially intact. The neoplasm assumes the shape either of a ring or a polypus; occasionally its formation resembles that of villi. Throughout the course the tumors are almost always of small size. Often their diameter does not amount to more than 1 cm., and in length they are comparatively short; some appear to attack the wall only to an extent of 5 cm. Nevertheless, tumors from the size of a walnut to that of a fist have been observed. Their concealed position and small size almost invariably make palpation of tumors of the choledochus impossible. In accordance with the well-known laws of localization for cancer in general, carcinomata of the bile passages are found chiefly in the most narrowed areas of the canal (at the mouth of the common bile duct, at the confluence of the cystic and hepatic duct, at the point of bifurcation of the hepatic duct). At the autopsy of my cases, 7 times a tumor was found in the vicinity of the diverticulum of Vater, or, at least, below the middle of the course of the common duct, which was about 8 cm. in length; in 3 cases carcinoma near the junction of the hepatic duct and the cystic, and once only in the hepatic duct. In another case, there was a contact carcinoma of the ductus choledochus conjoined to a primary tumor of the gall-bladder, in another a projecting cancer of the cardia ventriculi attached to the gastrohepatic ligament, and another of the common bile duct and cystic duct. In passing it may be remarked that metastatic cancerous nodules are also found isolated in the ductus choledochus. Clinically it is of little significance whether the carcinoma be primary or secondary, since the prognosis of a cancer of the gall-bladder, for example, one which by proliferating occludes the choledochus, is in any case still more serious than that of a primary tumor in the bile duct. The exact localization of the origin of the neoplasm, as may be readily understood, sometimes is difficult. In two of the seven cases of carcinoma of the ductus choledochus which have been referred to, the lowest portion, corresponding to the diverticulum of Vater, ruptured into the duodenum; in one there was simultaneous proliferation to the pancreas. Inversely, primary carcinoma of the duodenum may not

only include the mouth of the choledochus, but, invading deeply, may also occlude the bile duct. The ductus choledochus, below the insertion of the cystic duct, may also be occluded by the tumor mass which burrows through the latter to the gall-bladder, where, in the narrow portion which is immediately connected with the cystie duet, it may be of very small size. That the neoplasms actually originate in these small masses may be readily recognized from the fact that the secondary growth permeates the entire wall of the gall-bladder and also attacks the substance of the liver in its immediate vicinity. I have never seen a neoplasm of the cystic duct which was not connected with carcinoma of the gall-bladder or of the ductus choledochus (hepaticus). According to some statistics (Courvoisier), in 19 cases of cancer of the choledoehus the origin of the neoplasm was 9 times in the beginning of the duct near the junction of the cystic and hepatic duets, 3 times in the middle, 7 times in the pars intestinalis, that is, in the ostium. Carcinomata of the gall-ducts are evidently rarer than those of the gall-bladder. Among the causes of obstruction of the choledochus by tumors, carcinoma of the pancreas, from its frequency, can alone be considered. Etiologically, heredity here appears to play no rôle. Of my 13 cases 7 were males. The women who were attacked had almost all borne children (one 13 times). A predominant implication of the male sex has been frequently emphasized, and this preponderance perhaps had too much weight attached to it in comparison with the previously mentioned implication in cancer of the gall-bladder, in which disease females unquestionably show by far the greater number of cases. The importance of the question consists in the intimate relation of carcinoma of the gall-bladder to cholelithiasis. In opposition to the views of Naunyn, Leichtenstern, G. Hoppe-Seyler and others, this etiologic factor, however, quite apart from the consideration of the implication of either sex, does not appear to occur to quite the same extent in eancer of the ductus choledochus and hepaticus as the material at hand seems to warrant us in concluding. In the autopsy reports of my own cases, only once do we find mention of a stone in the biliary passages (gall-bladder) and once in the intestines (appendix). Positive proof of cholelithiasis could be determined at most in only three of the histories of my patients; in some of the cases in prior and more recent literature the stone was found directly in the region of the neoplasm, that is, in the carcinomatous structure. Twice in my cases abdominal trauma was assumed to be the cause of the disease. The frequent seat of carcinoma of the choledochus in the lowest portion of the duct appears to me at least to favor the view that deleterious action of the intestinal contents may be a cause. In regard to age, only one of my patients was under 50; seven were past 60, the oldest was 72. In no case was the anatomical extension of the disease limited to the biliary organs. Secondary metastatic carcinoma was found five times in the liver, twice in the lung, twice in the duodenum, once in the pancreas,

once in the kidney, once in the uterus, and frequently in the peritoneal glands. In regard to the implication of the proliferal lymph-glands, later reports must be used for comparison. In opposition to the general view that this rarely occurs on account of the absolute stasis of bile and the early death (Leichtenstern counts it among the greatest exceptions!), in my experience the development of metastases was quite invariable and profuse. The liver was always enlarged and its tissues coarser; well developed biliary cirrhosis developed in 3 cases. The portions of the ductus choledochus and ductus hepaticus above the obstruction showed, without exception, more or less extreme dilatation. In 7 cases the gall-bladder was markedly distended to the size of a fist. We find in literature some mention of biliary passages thus dilated from which, by puncture, almost a liter of fluid was obtained. In cases of decided enlargement of the gall-bladder, the tongue-like elongation of the liver, corresponding to the gall-bladder (Riedel's lobe), was noted. In 4 of my cases dilatation of the gall-bladder was not present, yet there was no apparent reason for this absence; twice in the autopsy reports the gall-bladder is mentioned as small and showing a simultaneous long-existing cholelithiasis and cholecystitis. Cholangitis (cholecystitis) with a purulent exudate was present in 4 cases; traces or remains of inflammation of the biliary passages were much more common. Diffuse purulent cholangitis of the finer branches of the hepatic duct (Ely) I have not seen. A few isolated cases of multiple abscess of the liver are mentioned in literature (van Gieson). The adhesions (and ulcerations) observed by me are of such a character as to require no detailed description. None of my patients succumbed directly to perforation, formation of fistula, or rupture of the biliary passages. Dilatation of the pancreatic duct and connective tissue proliferation in the head of the pancreas I saw repeatedly. In the majority of cases (even without other complications) the spleen was distinctly enlarged. Ascites as well as anasarca is mentioned expressly six times in the autopsy findings; in reality it is probably more frequent. Hemorrhage from the neoplasm itself I have observed only once; the hemorrhagic diathesis is mentioned in 4 cases. Appendicitis was diagnosticated in one case and the abscess was opened. At the autopsy the necrosis of the appendix was discovered to be due to a gall-stone (cholesterin). In 3 cases sepsis of the oral cavity (tonsillar abscess, parotitis) was a sequel.

Primary cancer of the duodenum is, as already stated, extremely rare (we are here only interested in that form with a *periampullar* seat; the *parapyloric* form (Nothnagel), that is, cancer situated above the diverticulum of Vater, I shall not describe at all). The former may develop upon the base of a peptic ulcer. This is either the round form of ulcer about the size of a silver quarter of a dollar with medullary infiltrated borders near the mouth of the ductus choledochus, or the same base, in the center of which a polypus-like protruding portion of mucous membrane is still pre-

served which carries the ostium of the duct. Such carcinomatous growths, naturally, do not form palpable tumors. The constriction of the ductus choledochus and Wirsung's duct, under these circumstances, is extreme. Extending further, the tumor mass may directly occlude the ducts. The choledochus is dilated to the size of the small intestine, the hepatic duct to the diameter of the thumb, and the gall-bladder attains the size of two fists. The duct of Wirsung is similarly dilated, so that it stands forth prominently, and the smaller lobes of the ducts resemble cysts. In contrast to this there are also cauliflower-like and submucous duodenal cancers, which, on account of their size, may be palpated during the life of the patient. These latter tumors are prone to necrosis and hemorrhage. As long as the growth does not rupture, there is occlusion of the ostia; later, the bile and pancreatic juice may find an outlet. The mouth of Wirsung's duct is dilated and surrounded by shreddy, secondary growths; the same is true of the mouth of the common bile duct. The biliary passages and the pancreatic duct appear to be only moderately dilated. The mesocolon is drawn toward the growths, the intestinal wall is perforated. The head of the pancreas is, as a rule, inseparably attached to the base of the growth.

A case of *cicatricial duodenal stenosis* which I have observed is as follows:

A day laborer, aged 40, who, since his 20th year, had suffered from digestive difficulty and vomiting. In October, 1896, he was suddenly seized with chills and spasmodic pains in the right hypochondrium, and immediately after there was a yellow discoloration of the skin as well as a brown color of the urine. After a brief period the jaundice cleared up. Two months later he had another attack, and at the end of December a third and severe attack lasting 10 days. Since the 2d of February, 1897, his condition has been stationary. He now has pain in the right hypochondrium; the feces show no bile. Occasionally there is vomiting. Fever (up to 102.2° F.) until the 12th of February; later it disappeared entirely. Marked emaciation. Extreme jaundice. No edema; abdomen occasionally shows meteorism. No collateral circulation. Ascites absent during the entire course. Liver is indurated, border readily palpated, surface smooth. Gall-bladder not palpable. Feces show no bile and are rich in fat. Urine contains profuse amounts of bile. Attacks of pain constantly recur.

March 10th, epistaxis. March 14th, right-sided pneumonia. March 17th, vomiting of brownish-black masses; sub-normal temperature. March 23rd, death.

Autopsy: Stenosis cicatricosa duodeni cum occlusione ostiorum d. choledochi et Wirsungiani accidente cholestasi et ictero. Perforatio vesicae fellcae supra hepar. Abscess. chron. choliferus. Dilatatio et hypertrophia ventriculi. Peritonitis diffusa.

The stricture had established itself at the site of an old *fistula from gall-stones.* Stones were still present in the common bile duct. This case illustrates drastically the natural cure of incarceration from stone.

DIAGNOSIS; DIFFERENTIAL DIAGNOSIS

After having studied the special pathology of circumscribed adhesive perihepatitis in the porta hepatis, and the affections of the periportal lymph-glands, carcinomata of the head of the pancreas, primary and secondary tumors of the biliary passages, as well as cancer of the duodenum, we shall now turn to the *differential diagnosis*; first, of occlusion of the ductus choledochus due to *stone* and to *tumor* (of the biliary passages themselves and of the neighboring organs). In enumerating and describing the individual diagnostic factors, I shall utilize the most recent literature of the subject (the earlier literature is of little value), and, on account of numerous contradictions, I shall base these remarks upon my own experience. A special classification of the differential symptoms is hardly possible, nor is it necessary; the simplest division appears to me to be indicated by the method of clinical examination. On account of the rarity of obstruction of the choledochus, not only the usual diagnostic factors must be considered, but the exceptions also must be carefully noted; we must guard against schematic force!

Let us begin with the *history*. In the diagnosis of cancer of the choledochus, Leichtenstern lays special stress upon the fact that the affection is prone to occur in *women*. This coincides with the reports of numerous authors, but even Courvoisier has disproven it. My own experiences (see above) also conflict with this statement. Cancer of the pancreas probably attacks men more frequently than women. Bohnstedt maintains that while those persons affected by incarcerated stones usually attribute their malady to a definite trauma which they have sustained, patients with cancer report nothing of the kind. This does not coincide with my experience. The majority of my patients with obstruction due to tumor have attributed the sudden appearance of the affection to some error in diet, a trauma, or something similar. Courvoisier has proposed a diagnostic law: *Preceding colic,* particularly if combined with jaundice, is sufficient evidence for assuming an occlusion of the ductus choledochus due to stone. The repeated and frequent appearance for years of one, usually *brief, attack of jaundice,* especially if it be accompanied by colic, or have preceded colic by a short time, is one of the most infallible signs of cholelithiasis. The previous passage of small stones occurs only in occlusion due to calculi. Colic was a symptom in 80 cases of stone in the common duct, and in 51 of the cases collected by Courvoisier; in 26 cases of preceding, repeated, brief attacks of jaundice, colic was noted 22 times; in 74 cases of obstruction of the choledochus due to other causes, colic was reported only 4 times (in two of these last mentioned cases the previous passage of calculi must be considered). Naunyn also declares it to be the rule that incarceration of stone is preceded by many attacks of colic.

According to my observation, typical chronic occlusion of the choledochus due to stone certainly develops after prolonged gastric difficulty (epigastric pressure, spasm of the stomach) sometimes lasting for years; attacks of pain without jaundice first occur, then well-developed colic with fever and violent vomiting, but still without jaundice. This is then followed by jaundice, the intensity of which varies decidedly, and which, after a longer or shorter period, may show complete intermissions. Chronic gall-stone jaundice, therefore, originates in an attack of colic. Colic recurs, but, with every attack, it becomes less severe, the pains decrease, and only chills and jaundice, or the latter alone, remain. Many patients declare that with the appearance of well-developed chills and of permanent jaundice the character of the pain changes. Exceptionally, however, the pain and the other signs of an attack of cholelithiasis in occlusion due to stone may be entirely absent (in Courvoisier's cases their absence is expressly mentioned 10 times), and jaundice may appear without any indication of colic. Various subjective symptoms may then have preceded, but this is not necessarily so. I saw a case (confirmed by autopsy, which will be again mentioned) in which the patient (Ru.), without prodromes, some time prior to his admission to the hospital, and in consequence of an error in diet, was attacked with vomiting, diarrhea and chills; this was followed in four days by moderate sensitiveness to pressure in the right hypochondrium and permanent jaundice. If we ask whether *pain,* and of what nature, usually accompanies the development of tumors leading to occlusion of the common gall-duct, Leichtenstern maintains that, as a rule, especially in cancer of the biliary passages, cholelithiasis (colic), which may, perhaps, have preceded for years, is a factor. This does not agree with the experience of other authors nor with my own. Undoubtedly there are cases of obstruction due to tumor in whose history, even during the entire course of the affection, pain was scarcely ever, if at all, present; that is, there was at most only tenderness on pressure in the gastrohepatic region. In my cases the absence of pain or attacks of pain only prior to the appearance of jaundice is mentioned in 8 cases; in the remaining course of the disease in 5 cases. Among these, unquestionably, there must have been cases of primary neoplasm of the ductus choledochus. Among my 24 cases cholecystolithiasis is proven by autopsy five times, and by typical attacks of colic (exclusive of actual tumors in the biliary passages) three times. Therefore, pain of a varying character is generally, as I believe, much more frequent than its entire absence, particularly at the height of the disease. If Kehr maintains that, in occlusion of tumors, colic is rare, much depends upon what is included under the term "colic." The intensity of these subjective difficulties, before and during the permanent jaundice, naturally varies greatly, and shows all the degrees between an indefinite, disagreeable sensation of pressure in the gastrohepatic region, and boring, lancinating and spasmodic pains of intolerable severity

which rob the patient of his sleep, make every position he may assume unbearable, compel him to cry aloud and to long for operative relief, etc. There are cases in which continuous pain predominates; in many others exacerbations are conspicuous, the pain comes in paroxysms, and the attacks are of varying duration. Not infrequently nausea is combined with the pain. An error in diet may produce the first, which is then followed by jaundice, and gastric difficulties not rarely introduce the series of symptoms; subsequently the paroxysms may follow meals. *Vomiting* (vomiting of bile) is mostly absent, that is, such as would correspond to typical gall-stone colic at the height of the attack. Severe, frequent vomiting, often bilious in character, which persists for long periods during the course, is invariably found in duodenal and also in pancreatic cancer when stenosis of the duodenum takes place. The *deep-seated, neuralgic pain, chiefly localized in the region between the pit of the stomach and the navel, radiating toward the back,* and usually referred to the *solar plexus,* is an important diagnostic factor only in certain cases (compare the history of the two cases quoted above). The pains arising from the biliary passages themselves also usually radiate to the gastric region, to the back, and especially to the shoulder. I am inclined to attach especial value to this *shoulder pain* in the diagnosis of affections in the region of the biliary passages, but I have never succeeded in making a positive differentiation between occlusion of the choledochus by a neoplasm of this duct or by a pancreatic cancer from this sign alone. I attach a certain significance to the circumstance that, in cancer of the duodenum, the pains increase after the ingestion of food. But this is a differentio-diagnostic factor of but relative importance, in that pains in obstruction due to tumors of the ductus choledochus only exceptionally correspond to typical colic (*pains and vomiting*) of gall-stone (in which, of course, I leave entirely out of consideration secondary carcinoma of the ductus choledochus in primary cancer of the gall-bladder).

In the diagnosis of cholelithiasis, Neusser and Ortner attach great importance to the *jaundice* which *in a brief time becomes marked,* that is, soon reaches its *maximum.* A villous cancer growing rapidly may also in a short time give rise to extreme icterus. In carcinoma of the duodenum, the appearance of jaundice is said to be much more gradual. In a series of cases of carcinomata of the pancreas Leichtenstern found that icterus appeared in an acute form and in a few days reached its maximum. I attach but little weight to the statements of the patient, which are often quite vague, as to the manner in which the permanent jaundice appeared. I know of undoubted cases of incarceration of stones in which jaundice developed gradually; this seems most apt to occur in cases in which the stone is partly in the cystic duct, and partly in the common duct. Among my own cases of obstruction due to tumor, 5 times the history showed that jaundice had appeared "suddenly."

in a few hours or in a night, and this was also true in cancer of the pancreas.

Another, perhaps significant, diagnostic difference between the development of permanent jaundice from obstruction due to calculi contrasted with occlusion from tumor is the *timely extension* of the same. This factor is naturally considered with the history, and in the further observation of the case. Here again Courvoisier was foremost with the definite statement that permanent jaundice lasting several years generally indicated stone and excluded any other form of obstruction. He mentions cases of obstruction due to stone lasting 2 to 5 years, and even longer. Kehr operated upon and cured a patient who, for 12 years, suffered from the symptoms of occlusion by stone. Schüppel, as well as Neusser and Ortner, on the other hand, exclude cancer in general (except, possibly, primary carcinoma of the gall-bladder) if the jaundice persists longer than 6 months. This latter period, however, appears to me to be entirely too limited. In my cases of primary cancer of the biliary passages, the total duration of the course from the onset of chronic jaundice up to the time of death was 7 to 9 months; in one case only was it less than 6 months; in my cases of cancer of the pancreas, 3 lasted less than 6 months, and in all the others the duration was 8 to 11 months; in duodenal cancer the course was somewhat longer than 6 months. Perhaps the average duration in cancer of the head of the pancreas is slightly less than in cancer of the choledochus. With Naunyn and others, I think it wiser to estimate the duration of jaundice which excludes cancer at about one year. In practice, however, there is no absolute, precise law. For, in occlusion due to stone, death may occur also after a brief duration of the jaundice, often a few months, often only weeks or days! In most of the known cases, death was preceded by symptoms of hepatic autointoxication from the 6th to the 12th month; therefore, chronic gall-stone jaundice rarely lasts longer than one year. The reason why occlusion of the common duct by a tumor produces jaundice which lasts such a short time is, according to Courvoisier, simply this,—that here the underlying disease in some other manner early causes death. But I do not believe this. I am inclined to agree with Kehr, and admit a chronic closure of the common duct by stone in the supraduodenal portion, and one due to calculus in the vicinity of the duodenal papilla. In the latter case, and under severe circumstances, the jaundice is at once intense, and varies but little from that observed in most obstructions due to carcinoma. In these later cases, however, provided a choledocho-duodenal fistula does not bring relief, or if a choledochotomy is not performed, a fatal termination is just as certain and as rapid as in carcinoma of the common duct and pancreas, and with the same symptoms of severe inanition and hepatargy. In no less than 14 of my own cases, without regard to the focus for the development of the cancer, this was absolutely demonstrated (often by prolonged coma with

or without the hemorrhagic diathesis). Among auxiliary causes of death suppuration was found in four cases, an old syphilis in one, portal vein stasis in another, and in a few, metastases.

Jaundice, as a cardinal symptom, occupies a leading position in the status and course of the clinical history. It may be assumed from the onset that a stone with its changeable form, or even a great number of stones, will not completely occlude the lumen of the bile ducts, as would, for instance, be the case with a soft tumor which perfectly adjusts itself to the lumen and the wall of the canal, and entirely occludes the former. Even Cruveilhier attempts to base this theory of constriction, *a priori,* upon the comparison of the pathologico-anatomical changes which occur in incarceration from stone. Behind the stone arrested in the common bile duct stasis of bile occurs, whereupon the passage dilates, the lumen increases to the circumference of a finger, or even that of the small intestine. Then the bile rises to the stone, floats in the dilated common duct, and passes it. The jaundice may then entirely disappear. But the stone invariably floats along to lower parts of the gall-duct toward the intestine. Clinical experience confirms the opinion that in chronic occlusion of the common bile duct by stone, a *decided variation in the intensity of the jaundice,* even its *complete disappearance,* is very characteristic. Under these circumstances the urine likewise shows variations in color between dark and light, and only in rare cases are the feces permanently free from bile. In cases of obstruction due to tumor, the jaundice is usually intense, and does not vary so manifestly. The feces are persistently acholic. In contrast to the usual conditions, however, we must bear in mind that also in incarceration from stone (in the papilla of the duodenum) the complete exclusion of bile from the intestine, with all its serious consequences (hepatic intoxication, the hemorrhagic diathesis, and death in from 6 to 12 months) is quite possible, and this may occasionally occur even when permanent jaundice has been the first alarming symptom of a cholelithiasis previously latent. An example of this kind is my case before referred to (Ru.). Here for a short time only the jaundice was moderate, the feces slightly tinged with bile, but the symptoms rapidly became more intense, and the feces subsequently and during the 7 months of the protracted course of the disease were absolutely acholic; the course was that severe form just indicated. Moreover, in my experience it is the rule that also in cases of obstruction due to tumor, variations in the intensity of the jaundice are quite manifest, but they are never very decided ones. Particularly at the onset of the affection, paroxysmal aggravation of all the subjective symptoms, produced by a still more marked impediment to the flow of bile, brings about a renewed staining of the visible membranes. Haarmann believed that a complete but temporary disappearance of jaundice in obstruction from tumor could be explained by the destruction, and its recurrence by the renewed proliferation, of a soft tumor

which in his case was situated at the ostium. Courvoisier admits the probability of similar conditions from analogous observations by Bourceret and Bradbury. Neusser and Ortner designate as a decisive factor in the diagnosis of carcinoma of the common bile duct the circumstance that, in the course of a jaundice which has developed to its maximum intensity combined with acholia of the feces (usually after a duration of from 2 to 3 weeks) a full, even abnormally profuse, biliary staining of the fæces may again occur; this may happen once or repeatedly, suddenly or at least rapidly, occasionally with fulminant symptoms; there may be a slight admixture of blood to the dejecta or to the vomitus; in a brief time, the condition gives place to a new period of acholia. I have reason to believe that instances of this kind frequently occur. In one of my own cases of primary cancer of the choledochus (at the onset of permanent jaundice) the previously intensely yellow discoloration of the skin decidedly diminished, and the white, clay-like stools again became tinged with bile; this change lasted for only 8 days, and was followed by an aggravation and permanent acholia which was unyielding. Hemorrhages from cancer of the common bile duct, even to a marked extent (black feces), are not uncommon. One of my cases of cancer of the duodenum also exhibited the condition described by Neusser, and an anatomical preparation satisfactorily explained it as due to decided ulceration. Naunyn also mentions a case of Coupland's of neoplasm in the duodenal wall at the diverticulum of Vater, in which a disappearance of the jaundice, biliary staining of the fæces, and a simultaneous decrease in the size of the distended gall-bladder could be repeatedly recognized. In two of my cases of occlusion of the choledochus from tumor of the pancreas, besides a manifest and very varying jaundice of the skin, there was long-continued diarrhea, and feces containing bile, some showing deficiency of bile, and completely acholic stools alternated. Finally, cases of obstruction from tumor exist (two among my own) in which the feces up to the time of death show only a feebly feculent character. Somewhat different from the variations in the intensity of the jaundice in incarceration from stone and occlusion from tumor, which have been described, is a clearing of the skin and urine after a long continuance of the jaundice without the fæces losing their clay color. The significance of this prognostically unfavorable sign is evident from the foregoing descriptions.

In the opinion of different investigators the diagnostic *importance of fever* varies. By many the presence of fever preceding and during the permanent jaundice, and especially the repeated occurrence of chills at regular or atypical periods, combined with attacks of pain, with sudden rises of temperature, and, later, also the remittent or continuous rises of temperature in chronic obstructive jaundice are referred either to gall-stones, that is, to suppuration, to ulceration, etc., in the biliary passages (the liver) or to a superadded pyemia. Courvoisier reports that in 80

cases of occlusion by stone 19 (therefore about $\frac{1}{4}$), and of 74 obstructions due to other causes 7 (about $\frac{1}{10}$), were accompanied by fever. I must emphasize here that I also know of cases of impacted stone which, in certain phases of the affection, showed jaundice only some time after the attacks of fever (previously frequent) had ceased entirely. Leichtenstern maintains that as soon as cancer of the gall-bladder or of the biliary passages appears the preceding inflammatory phenomena due to gall-stones cease entirely. High fever or chills are said categorically to contraindicate the presence of cancer although numerous other symptoms may be present which favor it. According to Kehr, in occlusion by tumor fever occurs in exceptional cases, particularly in the last stages of carcinoma. In the case of scirrhus of the pancreas this author has observed multiple abscesses in the liver and perihepatic suppuration. Naunyn emphasizes the fact that fever combined with chills is observed with especial frequency in neoplasms of the liver (even those due to metastases). In 15 of my cases of occlusion due to carcinoma, there was fever lasting a long or short time. Only 5 times, either clinically or at the autopsy, was it possible to attribute it to a complication outside of the biliary passages themselves (parotitis, tonsillar abscess and pyemia, cystopyelitis, suppuration of the glands of the neck, gall-stone appendicitis). Fever, with either a moderate, intermittent or remittent course or (not so frequently) with decided rises in temperature to 101.3° F. and 103.1° F., was present. In the late stages of the disease (leaving out of consideration the closing days of life, when lobular pneumonias, etc., developed) the fever usually but not invariably became more marked. Periodically recurring or even isolated chills I have never observed (except in a case of lithogenous cancer of the gall-bladder which proliferated into the biliary passages). I am unable to decide whether fever is more frequent in that form in which there is proliferation into the duodenum leading to destruction, which is particularly the case in carcinoma of the head of the pancreas, or in primary cancer of the biliary passages. As will be observed, I can take only a middle position in regard to the differentio-diagnostic value of fever; all the more so since, in literature, I note that chills also occur in occlusion due to carcinoma.

The *degree of inanition* and the *loss of weight* in all of my cases of tumor obstruction have been very considerable; *cachexia* developed with exceeding rapidity. I have not observed that cachexia præcox is peculiar to cancer of the pancreas; nor have I noted subnormal temperature in such cases (the syndrome of Bard and Pic). In my cases the N-excretion in comparison to the intake was not determined. In doubtful cases, among other signs, light was occasionally shed on the diagnosis by the estimation of N and NaCl in the urine (diminished excretion of chlorin in cancer). I have no personal experience of the proportions of N and P_2O_5 in the excretion of persons suffering from obstruction of the choledochus by tumor, nor in regard to the increase of ammonia and the separation of

the total nitrogen into the various N-containing products of the urine. In all of my cases of obstruction by tumor in which an examination was made, deviations from the normal blood were noted, although not very decided: decrease of hemoglobin to 75–65 per cent. (Fleischl), decrease of the red blood-corpuscles to 5,800,000, 4,100,000, or 3,900,000; the number of white corpuscles (not directly due to the fever) varied between 5,800 and 16,400. In one case (carcinoma of the pancreas with enteritis and fever) very decided oxyphilia of the blood was observed. Finally, we should be most cautious in drawing far-reaching differentio-diagnostic conclusions from the miserable appearance of the patient and his loss of weight!

In discussing biliary stasis due to obstruction of the choledochus I have not minutely considered the dilatation of the biliary canal. In the first place, experience shows that dilatation occurs in the common and hepatic ducts. Particularly in cases of closure near the opening of the ducts into the intestine do dilatation and elongation occur, and these ducts may enlarge to the size of a thumb or a coil of the small intestine. The common duct occasionally forms a sort of cyst, protruding from under the liver, containing from one to several liters of bile, and has, in fact, been confounded with the gall-bladder upon which operation has been attempted. The dilatation of the final branches of the hepatic duct extends usually to the periphery of the liver, and may produce fluctuating protuberances on the surface of this organ, or, upon a uniform dilatation of the intrahepatic canals, the liver *in toto* becomes enlarged, but, as a rule, it shortly after again decreases in size. In chronic obstruction of the common duct *dilatation of the gall-bladder* and of the cystic duct does not occur with the same frequency. As we were inclined to believe from the reports mentioned in text-books, Courvoisier found the tissue changes in the gall-bladder different and more complicated. Previously it was the general opinion that permanent closure of the choledochus invariably led also to a dilatation of the gall-bladder. Courvoisier found, however, that among 109 fully described cases of dilatation of the gall-bladder, 17 only were due to occlusion by stone; all other obstructions of the ductus choledochus were due to different causes (10 to tumors in the interior, 8 to obliterations, 74 to external compressions). Furthermore, the dilatation of the gall-bladder in obstruction from stones was usually of only moderate extent, and in but a few cases was the organ stated to be enormously enlarged. On the other hand, dilatation due to other causes of occlusion was often very decided, and in some cases colossal tumors were formed. In contrast to these dilatations are 78 cases also collected by Courvoisier, in which, in spite of occlusion of the common duct and a patulous cystic duct, the gall-bladder was not enlarged but atrophic. Of these, 70 (i. e., 90 per cent.) were cases of impaction of stone in the common duct, 8 only (i. e., 10 per cent.) were due to other causes (in 5 of the last-mentioned

cases the gall-bladder also contained stones). Courvoisier separates his 187 cases into two groups, one containing 87 cases due to obstruction from stone and 100 from other causes of occlusion. With incarceration of calculi, atrophy of the gall-bladder occurred in 80.4 per cent., dilatation, which was found in 19.6 per cent. of the cases, being much rarer. The other causes of obstruction, on the contrary, much less often ran their course with atrophy (8 per cent.), dilatation being much more frequently present (92 per cent.). In impaction of stone in the common duct, therefore, dilatation of the gall-bladder is rare, the organ being previously contracted by connective tissue (chronic cholecystitis, atrophic effect of adhesion). In occlusion of the choledochus of other nature, however, dilatation is the usual result. Courvoisier collected 35 cases of obstruction of the common duct in which operation had been performed; in 17 cases due to stone 4 only showed dilatation, and in 18 due to pressure from tumor, from stricture, etc., dilatation was found in 16; of the first 17 there were 13, and of the last-mentioned 18 only 2, without enlargement of the gall-bladder. Of the 20 cases of dilatation of the gall-bladder, 4 only were due to obstruction by stone, 16 being from other causes. The correctness of this view has been confirmed in a large number of cases by subsequent investigation. Encklin (1896), in a total of 172 cases of occlusion due to stone, found contracted gall-bladder present in 64 per cent.; in 19.7 per cent. the organ had retained its normal size, and in only 16.25 per cent. was it dilated. On the contrary, in 129 cases of occlusion from other causes (stricture of the choledochus, compression of the duct by enlarged lymph-glands, tumors of the common duct and of the pancreas) the gall-bladder was found dilated in 87 per cent., and contracted or normal in only 6.5 per cent. Courvoisier regarded this incontestable contraction of the gall-bladder from the presence of stone in the common duct a particularly valuable point of support in the differential diagnosis. Naunyn, Kehr, Langenbuch, Bohnstedt, and others concurred with him. This diagnostic point is referred to (particularly in French literature) as *Courvoisier-Terrier's symptom*. Terrier, however, only followed Courvoisier's teaching. G. Hoppe-Seyler maintains a conservative position. Leichtenstern, "naturally," does not conform to the Courvoisier rule in deciding the question whether occlusion of the common duct due to stone or cancer of the biliary passages is present, the latter of which he declares to be lithogenous; he maintains absolutely that in primary carcinoma of the choledochus there is no enlargement of the gall-bladder, while, in contrast to this, in cancer of the pancreas a well-filled gall-bladder can be determined. Neusser and Ortner declare in the diagnosis of primary cancer of the ductus choledochus that a distinctly enlarged gall-bladder is of "little importance." Quénu also maintains that the Courvoisier system is not absolutely decisive, and reports some mistakes in diagnosis.

I have already briefly alluded to the results obtained in my autopsies. In my opinion, in a diagnostic respect (I am considering only the diagnosis prior to operation), and without weighing the autopsy findings, the question is as follows: Are the cases with a non-palpable gall-bladder in obstruction due to tumor, and the cases with an enlarged palpable gall-bladder in occlusion due to stone, actually so rare that Courvoisier's sign may be looked upon as pathognomonic, and that, in each individual case, a decision may be made from this? In my 8 cases of primary carcinoma of the choledochus, during the entire course of the disease the gall-bladder was 4 times non-palpable, and in my 7 cases of cancer of the head of the pancreas with occlusion of the common duct, this was the case 3 times. In one instance of primary cancer of the gall-bladder with subsequent cancer of the choledochus, the gall-bladder could not be felt. In one case of duodenal carcinoma the gall-bladder was visible, in the second case it could never be seen. The gall-bladder was observed to be anatomically dilated in some cases in which the enlargement could not be discerned clinically. The largest tumor of the gall-bladder which was found during life in my cases was about the size of an apple. The tumor invariably had the characteristics of the tumors of the gall-bladder which have been described. Fluctuation at the top of the tumor was never absent. In two cases with particularly well-developed Riedel's lobes it was readily perceived that a neoplasm had formed at the point of transition into the coarser liver. In a clinical history pain upon pressure of the tumor is sometimes emphasized, sometimes absent. The growth of the tumor and its variation in size have been occasionally mentioned. In 4 positive cases of obstruction due to stone I remember that the gall-bladder was more or less enlarged to palpation. In one of these cases, we were able during the life of the patient to determine that the gall-bladder was not absolutely smooth and freely movable, as is the rule in occlusion due to carcinoma. In my opinion, in cholelithiasis much depends upon how and when the impaction of stone takes place. In a case repeatedly mentioned (Ru.), in which permanent jaundice was the first alarming symptom of cholelithiasis, and the cystic duct was not obliterated, the gall-bladder was very markedly dilated, yet no stone was present in the cystic duct, and, therefore, in addition to the obstruction of the choledochus, there was neither dropsy, empyema of the gall-bladder, nor obliteration of the duct. Courvoisier collected 12 cases from literature in which the last-named accident supervened. Kehr also calls attention to the diagnostic difficulties when we have a combination of dropsy or purulent cholecystitis of the gall-bladder with chronic occlusion of the choledochus. He operated on 5 cases of this kind. In the differential diagnosis between occlusion of the choledochus due to stone and to tumor, after all that has been said, and apart from the fact that the exceptions also must be considered (on account of the rarity of the cases) no actual pathognomonic importance

can be attributed to the Courvoisier symptom, the value of which, I believe, is often exaggerated.

According to Naunyn, there is often no marked *enlargement of the liver* in chronic gall-stone jaundice. With obstructing carcinoma of the common duct and of the pancreas, also, the liver is usually small (for cancer of the gall-bladder, Bard and Pic hold similar views), while in cancer of the gall-bladder, as a rule, decided enlargement and nodular consistence of the liver are never long absent. In my clinical histories of tumor occlusion, enlargement of the liver is frequently and particularly emphasized. In 5 cases, metastatic cancerous nodules could be palpated on the surface of the liver during the life of the patient; anatomically, metastases of this organ have been even more frequently proven; upon examination, the liver may appear smooth although carcinomatous nodules have already formed. Considering our previous remarks on this subject a minute examination of the liver for secondary cancerous nodules (and also palpation of the cavum Douglasii) should never be omitted. When carcinoma of the liver is absent, the consistence of the organ is, as a rule, moderately increased. Biliary cirrhosis was twice anatomically determined with certainty during the life of the patient.

Most authors agree that, in the majority of cases, the *tumor* which causes obstruction, as such, notwithstanding exact and repeated investigations, is not palpable. I was able to palpate it in only one of my cases of carcinoma of the pancreas and in one of my two cases of cancer of the duodenum (the latter only occasionally). Leichtenstern describes a tumor at the head of the pancreas which may generally be definitely palpated; carcinoma of the duodenum below the papilla he diagnosticates by the presence of a tumor, etc. Moncorgé emphasizes the presence of a tumor in the median line. In the case of tumors of the pancreas leading to obstructive jaundice, I was unable to confirm this. In malignant tumors which develop in an area insusceptible to palpation, G. Hoppe-Seyler expects results from the X-rays; but I cannot share this hope because of my experience in other cases.

The consensus of opinion (Naunyn, Kehr) is, that *ascites* is absent in uncomplicated jaundice due to stone. In my case (Ru.) it was present as well as anasarca and biliary cirrhosis, without peritonitis and without thrombosis of the portal vein. On the other hand, in neoplasms of the biliary canals and in carcinoma of the pancreas, ascites is not rare. Excepting one case of cancer of the pancreas in which aspiration was necessary, I found slight ascites 10 times, and anasarca 6 times.

Naunyn reports that *splenic tumor* is rare in carcinoma; on the other hand it is common in chronic gall-stone jaundice (not merely in addition to diffuse hepatitis!). In 8 of my cases of obstruction due to tumor there was decided prominence of the spleen, and in 4 it could be palpated. I do not believe the condition of the spleen of paramount significance in

a differential diagnosis. According to my experience, in any chronic jaundice (even in catarrhal jaundice apart from cirrhosis), the spleen may be enlarged; and, moreover, the enlargement of the spleen may also be explained by a superadded stasis of the portal vein (pressure of the tumor upon the trunk or branches).

According to Leichtenstern, a sign which is of great importance in the diagnosis of cancer, although not very frequent, is that the malignant infiltration causes a *swelling of the "jugular gland"* (Virchow's gland), a lymph-gland situated behind the clavicular insertion of the sterno-cleido-mastoid. Laterally this swelling may often extend above the muscle, may attain the size of a cherry, a chestnut, or, occasionally, of a hen's egg; it is invariably present on one side, generally the left, is occasionally isolated, i. e., with no other enlargement of glands in the supraclavicular region or in the neck. Kehr confirms Leichtenstern's observations; but the gland is found only in advanced stages of the disease when the diagnosis is no longer difficult. My clinical histories contain exact reports of the enlargement of peripheral lymph-glands; frequently of the inguinal glands, sometimes those of the axilla (particularly upon the right side); in the neck, as a rule, the enlargement is multiple and bilateral, and in the supraclavicular regions; enlargement of the jugular gland alone is mentioned rarely; in one case it was decidedly increased in size and finally broke down.

In the preceding, a series of factors are mentioned which are of varying importance, which, carefully combined and utilized, will facilitate the differential diagnosis between chronic impaction of stone and obstruction due to tumor. Incarceration of stone complicated by carcinoma of the common duct (lithogenous) which occurs occasionally (see above), scarcely permits a positive decision; in such cases, from the standpoint of professional treatment only the diagnosis of cancer is necessary. Stricture of the common duct following cholelithiasis is not always characterized clinically in the picture of the disease. In such cases, on the assumption that stones are present, I advise operation, and in case no stones are found, operative procedure is to be limited to the breaking up of adhesions, etc., which is of marked, although only temporary, benefit. Complete obliteration of the choledochus, provided intense permanent jaundice (with enlarged or non-palpable gall-bladder), repeated attacks of jaundice of brief duration, or typical colic with fever have not preceded, may be referred to cholelithiasis; and in a differentio-diagnostic respect, ulcer of the duodenum (cicatricial stenosis of the duodenum) and obstruction due to cancer must be principally considered.

Up to the present time, the only diagrammatic methods for the grouping of the different forms of occlusion of the biliary passages not due to stone have been short formulas proposed by Neusser and Leichtenstern; Nothnagel also furnishes such for the duodenum, Oser for cancer of the pancreas.

SYMPTOMS IN FAVOR OF PRIMARY CANCER OF THE COMMON GALL-DUCT

According to Neusser

A duration of the disease beyond six months excludes cancer. Decisive negative signs: Absence of tumor upon palpation. Positive factors: Suddenly during permanent jaundice and while there is an admixture of blood to the feces, complete biliary staining of the feces is seen, rapidly changing again to acholia. Eventually infectious cholangitis. Prolonged absence of gastric symptoms. Less important: Distinct enlargement of gall-bladder.

According to Leichtenstern

Aged people, particularly women. Cholelithiasis, even though years previously. No palpable tumor. Metastases extraordinarily rare. No fever. Painless melasieterus of long duration. Acholia of the feces. Liver large and smooth. No tumor of the gall-bladder. Cachexia. Anasarca. Eventually: Enlargement of Virchow's gland.

SYMPTOMS IN FAVOR OF CARCINOMA OF THE PANCREAS AS A CAUSE OF OCCLUSION OF THE COMMON BILE DUCT

According to Neusser

Besides jaundice: Celiac neuralgia; from the onset decided aortic pulsation in the epigastrium, and the absence of a localized, or at most an indistinctly palpable, tumor. Later glycosuria (maltosuria), the absence of indicanuria; profuse amounts of transverse striped muscular fibers; undigested starches, deficiency of fat-splitting in the feces.

According to Leichtenstern

Intense jaundice, acholia of the feces. Occasionally a tensely filled gall-bladder. Pancreatic tumor usually distinctly palpable. Persistent and severe gastric symptoms. Late disappearance of free hydrochloric acid from the gastric contents.

SYMPTOMS IN FAVOR OF CARCINOMA OF THE SMALL INTESTINE BELOW THE DIVERTICULUM OF VATER

According to Neusser

Jaundice; acholia of the stools, temporarily disappearing and then recurring; a gradual change in, perhaps even gradual first development of, the jaundice. No marked symptoms of pyloric intestinal stenosis, but other dyspeptic symptoms without recognizable disturbances of the gastric chemism. Melena without hematemesis. Late: Lumbar pains. Slow course. Carcinomatous metastases often absent (liver).

According to Leichtenstern

Rarity of this carcinoma. Duodenal cancer early causes stenosis of the intestine, in consequence of which massive bilious vomiting and gastrectasis occur. Tumor. Conspicuously acute development of the jaundice.

In regard to my own views, in several points I must agree, and very frequently disagree, with these contradictory statements, and my position may readily be determined from the foregoing explanations. One point only I shall briefly touch upon. In regard to the presence of gastrectasis, which is maintained by Leube in those cases of duodenal cancer in which the neoplasm is situated in the diverticulum of Vater, I concur in the opinion of Neusser. Typical, graphite-like fatty stools, i. e., such as are about one-half composed of fatty acid needles, are mentioned in only one of my cases. In regard to fat-splitting in the intestine, I, unfortunately, undertook no special investigations. As, however, according to experience, in carcinomata situated at the mouth of the ductus choledochus there is sometimes a simultaneous occlusion of the duct of Wirsung, and in consequence an exclusion of the pancreatic juice from the intestine leading to dilatation of the pancreatic duct and even to connective tissue proliferation in the tissue of the pancreas (I saw this in two cases), I do not expect such examination of the feces to aid us much in the differential diagnosis. In all of my cases there was either constipation or diarrhea. Glycosuria upon ordinary diet I did not once observe in my cases of carcinoma of the pancreas. In my recent cases, including the cases of cancer of the pancreas, the systematic test of the patient's power of assimilation for grape sugar never showed alimentary glycosuria beyond the ordinary amount; in only one case was there marked glycosuria with other signs of diabetes even on an albumin diet. This was a positive case of carcinoma of the ductus choledochus, with a metastatic, malignant involvement of the lymph-glands in the concavity of the celiac plexus, and slight hardening of the pancreas. Indoxyl was sometimes present in the urine, sometimes absent, but not only in the pancreatic tumors. Polyuria was an inconstant finding in my cases of cancer of the pancreas. Finally, but little reliance can be placed, diagnostically, upon the secretory function of the stomach: in two instances of cancer of the biliary passages, and in one of cancer of the pancreas, free hydrochloric acid was absent in the gastric contents. In one of the cases of duodenal cancer the reaction for free HCl was positive. The motor function of the stomach was not conspicuously impaired.

In conclusion, a few words concerning the differential diagnosis in the case of *the new-born*. Here intense jaundice, lasting longer than 14 days, with absence of bile from the feces, points to a serious disease of the large gall-duct. It cannot be merely a case of syphilis of the liver, but is due to the congenital absence, that is, fetal occlusion, of the biliary passages.

Our reason for making an early diagnosis minutely in cases of chronic obstructive jaundice is already indicated, but it is by no means, as Bohnstedt thinks, because, with the early recognition of a cancer of the biliary passages, this may be removed by operation. On account of the anatomical

relations and the condition of the patient, a radical cure is always excluded. No reports are known to me of successful extirpation of cancer of the choledochus, and the tumor situated at the head of the pancreas is particularly difficult to remove on account of its nearness to the duodenum and the excretory ducts which must be retained. Cholecystenterostomy, according to my own experience and that of others, I believe to be hopeless, for even successful anastomoses between the biliary passages and the intestine scarcely prolong the life of the patient beyond days or weeks. Anesthesia and its consequenees, and an exploratory laparotomy, are very badly borne by patients suffering from obstruction due to cancer. The results of these palliative operations in two of my own cases proved their uselessness to me more conclusively than the reports of Reclus, Regnier, Terrier, Socin and others. Therefore, while all operative interference is to little purpose (and is usually undertaken at the urgent request of the patient, solatii causa), every practitioner to whom the dispute between the internal clinician and the surgeon as to their special realm is an artificial one, will, at the present time, agree with those surgeons who in chronic obstruction due to stone perform choledoehotomy according to definite indications. For although cases of chronic gall-stone jaundice may recover naturally (choledochus-duodenal fistula) even after months (or years), unquestionably the danger in an expectant treatment is greater than from operative interference. But, not to compromise this operation unnecessarily, obstruction due to tumor must be entirely excluded from among operable cases.

3. NEOPLASMS OF THE LIVER

CLINICAL FORMS

The neoplasms of the liver itself which are important to the practitioner are *malignant* in the overwhelming majority of instances. Among these we deal mostly with *epitheliomata*; *sarcomata* are much rarer. The *benign* tumors usually mentioned are: *angioma, lymphangioma cavernosum, fibroma,* and *non-parasitic cysts.*

Most *fibromata* are not dangerous to life and cause no difficulty. Quincke, however, mentions a case in a child in which jaundice, acholia of the feces, enlargement of the circumference of the abdomen, a large, hard tumor of the right lobe of the liver, demonstrable dilatation of the gall-bladder and fever were observed; finally death occurred after a duration of the disease for 9 months. Larger fibromata were also seen by Chiari and Lanceraux. In the latter's case the neoplasm was multiple.

It is at least conceivable that, under particularly favorable circumstances, and when the diagnosis has been made early (perhaps by the aid of an exploratory laparotomy) the fibroma may be removed by operation. Sklifosowski (1890) successfully removed a fibromyoma, lipomatodes pe-

diculated, the size of a man's head, which was supposed to be a tumor of the omentum. When the tumor is centrally situated at the time of diagnosis, the conditions for the removal of the neoplasm are, as a rule, not nearly so favorable.

In spite of the fact, everywhere emphasized, that angiomata have no clinical importance, it nevertheless occurs now and then that, by the development of such a tumor in the liver, changes in its size and volume take place. During the life of the patient the diagnosis is only possible by exploratory laparotomy if, on account of a large tumor due to cavernous formation, which is rare, serious conditions are produced which require operative interference. Here we are principally concerned with the rare congenital form, which, according to Virchow, depends upon hypergenesis and dilatation of the hepatic vessels, and, according to Pilliet, presents fetal inclusions of meso-chymic tissue similar to the nævi vasculosi of the skin and the fissured angiomata. Steffen describes a solitary angioma, the size of an apple, in the right lobe of a child. Schuh and Maier saw two similar structures. Chervinsky found in the markedly enlarged liver of a girl 7 months old, such enormous cavernous tumors that the upper surface of the organ was covered with nodular prominences, similar to the condition in carcinoma. In this class probably belong the cases of v. Eiselsberg, Rosenthal and Hagedorn. The first author extirpated from a woman, aged 56, a carcinoma situated upon the border of the liver of the size of two fists which had distressed the patient for fifteen years. The case was one of slowly but steadily growing tumor in the right hypochondrium; it was occasionally very sensitive and hard to the touch; it had a small, nodular surface which could be somewhat displaced laterally, and which was connected with the liver by a demonstrable bridge. Prior to the successful operation it was questionable whether it was a tumor of the liver or of the kidney. Monneret and Fleury are, therefore, not correct when they report that angiomata which are susceptible to palpation differ from carcinomatous nodules by softness and elasticity, while indistinct fluctuation and the absence of the hydatid thrill permit a differentiation from abscess and from echinococcus cysts. Rosenthal's patient, aged 41, had observed a tumor in the abdomen for only a few weeks, and had become debilitated. A hard, slightly movable tumor about the size of a child's head, situated below the navel, and the connection of which with the liver could not be determined, was observed; laparotomy, however, showed that it was joined to Spiegel's lobe. The hepatic angioma, partially removed by Hagedorn, filled the abdominal cavity, having about the circumference of a gravid uterus. Only such isolated tumors as the last named (which are, besides, encapsulated by connective tissue) may be reckoned among those to be treated surgically with success. In considering the prognosis it may be briefly remarked that, according to Hanot and Gilbert, there are cavernomata which become infiltrated with black pig-

ment (melanoangiomata); this, however, does not permit the assumption that a tumor of this kind, or angiomata in general, will become carcinomatous.

Non-parasitic cysts of the liver must be included with the neoplasms because, at least in part, they originate from new structures, newly formed biliary passages; they may all be regarded as originating from the vasa aberrantia or from the intrahepatic biliary passages (related to the adenomata). We usually differentiate solitary, congenital, isolated cysts from multiple cysts of the liver developing during postuterine life. The congenital cysts, as a rule, do not reach great size; in certain cases, however, they become important on account of their decided volume, that is, in consequence of the ascites they produce, or by an obstruction to the birth of the child in question (Witzel, Bagot, Sänger, and Klopp). Multiple cystomata or cystic liver (cystic degeneration of the liver and kidney) may frequently but not invariably be combined with cirrhosis of the organ (also being in causal connection [?]). Besides those the size of a pin's head, there are others which contain as much as a liter of fluid; upon section, a liver of this kind shows a collection of cavities, the smallest of the size of a pin's head, and upon the surface of the organ larger and smaller protuberances form. The liver varies in size; usually it is enlarged. Biliary, portal vein, and even vena cava stasis may be the consequence. In combination with those of cystic liver, analogous changes occur also in the kidneys, to which the symptoms of chronic nephritis may be added. More rarely there are simultaneous cysts of the ovary, the uterus, the seminal vesicles, and of the thyreoid gland. Occasionally, multiple cysts of the liver cause no symptoms during the life of the patient. Sometimes such a liver may be palpated (nodular surface of the organ, fluctuation of the cysts). Puncture of the latter for diagnostic purposes should never be performed, as this induces suppuration; besides, it furnishes no positive facts. The rarer, simpler (that is, isolated) serous or biliary cysts of the liver usually form in advanced age (after the 40th year of life), sometimes earlier than this, and, as it appears, more frequently in women. Their seat is usually the upper, occasionally, however, the lower surface of the liver, immediately beneath the capsule. Their shape is globular or oval, but there is a variation in size which may even attain that of a child's head. The liver may be distinctly nodular, and sometimes the greater part of the cystoma extends above the hepatic mass; in rare cases the cyst may develop at the expense of the parenchyma to such an extent that the capsule and scant remains of tissue form only a thin sac. Suppuration has been observed. Operation upon such hepatic cysts (the cases in question are congenital cystomata and cystic degeneration) have been performed by Hueter, North, Terillon, Winckler, Kaltenbach, Berg, Bayer, and others.

In passing to the *malignant* neoplasms, we must first devote a few

words to *sarcomata* which up to the present time have unfortunately possessed greater pathologico-anatomical than practical interest. The occurrence of primary sarcomata was for a time denied; but some quite positive observations (Podroužek) have been recently reported. The focus of the disease is principally the region of the branches of the portal vein, and particularly the wall of the smallest vessels, as these are most apt to show the characteristic proliferation of sarcomatous tissue. At the autopsy of an undoubted case of Kahlden's which may be positively looked upon as primary, in a man, aged 32, who had suffered from ascites, the liver was found permeated by circumscribed, multiple, medullary nodules, the surface of which in many areas was umbilicated, so that they closely resembled malignant metastases. Microscopic examination revealed their size, the older cirrhotic changes, the sarcomatous nature of the tumors, and permitted the recognition of a distinct tendency to alveolar structure. The number of cell forms, as shown by the microscopic examination, also in sarcoma of the liver (primary and secondary), is a very varying one. Clinically, the question of the cell type of sarcoma of the liver is scarcely of importance; the malignity of the change shows only slight gradations. *Melanosarcoma,* although very rarely, also occurs primarily in the liver (Block, Belin and others). But the majority of sarcomata of the liver are, as a rule, secondary growths, and, as is readily conceivable, metastatic sarcomata of the liver are not as frequent as those of the lungs; why this condition is reversed in carcinomata, we shall see later on. Often the primary sarcoma which secondarily infects the liver is situated in an area of the body which may be reached by the physician (in the muscles, bones, breast, etc.); sometimes, however, a careful autopsy shows only a small primary tumor situated, for example, in the apple of the eye. In the case of melanomata the original neoplasm is usually in the choroid or in the skin (sarcomatously degenerated nævi pigmentosi). After the enucleation of the diseased bulb, years may sometimes pass without the liver being attacked. The fact is of practical significance that the skin may also be secondarily diseased. All secondary sarcomata in the liver cause either an extensive formation of nodular tumors or a more diffuse infiltration. The particularly malignant melanomata may be the cause of extremely large tumors of the liver, by far exceeding in size the non-pigmented growth. Occlusion of the capillaries and succeeding thrombosis of the intrahepatic portal branches produce ascites.

Excepting for the longer period of time which, at least occasionally, elapses between the appearance of the primary neoplasm and the discovery of sarcomatous metastases of the liver, the *symptomatology* of secondary sarcoma of the liver hardly differs from that of medullary cancer, that is, of secondary carcinoma of the liver. The sarcomatous metastases of the liver may remain latent during the life of the patient or may become prominent in the symptomatology. The organ enlarges decidedly, its sur-

face is covered with nodules, ascites appears (or may also be absent). Digestive disturbances, pain, and jaundice are frequent.

Melanomata of the liver are sometimes unrecognized in that they are masked by disturbances due to other and more prominent metastases. In other cases the liver affection is the most obvious, that is, it represents the only secondary disease. Then there are digestive disturbances, even vomiting, the strength decreases, the right hypochondrium is markedly sensitive to pressure, dull or sharp pain, increasing in paroxysms, and radiating to wide extent, appears; the volume of the liver increases decidedly. Its surface is by no means always nodulated; it is probably so only in the majority of cases; often it is smooth to the touch, and hard as wood. The right hypochondrium, the epigastrium, and, eventually, the entire abdomen are correspondingly distended. Meteorism and ascites are rarely absent; the spleen, however, is not decidedly increased in size. The stools may be acholic or may contain bile. Jaundice is very rarely present. Moderate fever is comparatively frequent. In all forms of hepatic sarcomatosis this latter symptom may be most prominent; its intensity is then considerable, and, with an intermittent type, chills, etc., may be added. The urine which, upon casual examination, is of normal color, if placed in the light, with an addition of saltpeter or chromic acid, turns black (Eiselt). This melanuria is of great diagnostic significance; unfortunately, however, the symptom is rarely present. The total duration of the disease after the affection of the liver has been determined varies between 1 and 6 months; on the average it is 2 to 3 months.

In by far the majority of cases, even with a tentative diagnosis, for the confirmation of which an exploratory incision may sometimes be necessary, the affection of the liver is so diffuse or manifold that (apart from other metastases of the neoplasm) operative removal cannot be attempted. The question whether a "malignant tumor" in the liver is solitary or multiple can only be answered after the abdominal cavity has been opened. Isolated resections of liver sarcomata have been reported (J. Israel and others).

Tumors of the liver composed of epithelial elements are still the subject of controversy among pathological anatomists, and particularly the relation between *adenoma* and *carcinoma,* although, according to the investigations of Hanot and Gilbert as well as of Siegenbeek van Heukelom, it is very likely that there is a fundamental difference between them; for the present, at least, the rare *solitary tubulous adenoma* of the liver is of special clinical interest because large tumors of this kind have repeatedly been successfully removed by operation. While I shall omit, therefore, the *benign, solitary, nodular hyperplasia* of the liver (Rokitansky, Klob, Mohamed and others), I shall briefly discuss the *multiple, nodular hyperplasia* (which, according to experience, has a certain tendency to adenomatous and carcinomatous formation) as well as the *multiple,*

tubular adenomata which I place with the group of hepatic carcinomata (cirrhosis carcinomatosa).

In a case of Keen's an adenoma which was extirpated proved to be a coccidia tumor; perhaps this etiology really plays a rôle (?). In rabbits, at least, there are adenoma cysts arising epidemically in the liver in the papillary proliferations of which Felsenthal and Stumm have found coccidia in various stages of development. As a rule, the solitary hepatic adenoma of man runs a comparatively benign course and does not give rise to metastases. In a few cases, however, the proliferation of the tumor into the portal veins and a thrombus-like growth has been described; in a case of Perls, an actual metastasis with glandular tubules was found in the sella turcica, and from the lumen of the tubules a greenish mass was excreted (Schmidt describes a primary hepatic cancer with metastases, and a nodule in the sternum of similar structure to that of the liver, in which bile was positively secreted); in another case secondary nodules were found in the lungs and in the mediastinal glands. As the hepatic adenomata increase in size, regressive metamorphosis and hemorrhage occur (with the formation of "blood cysts"), occasionally even to suppuration. In literature *cystoadenomata* are reported with a single layer of cylindrical epithelium and serum-like or colloid contents; they are connected with the biliary passages (compare what has been stated regarding cysts of the liver).

The previously mentioned regressive metamorphosis and the malignancy occasionally observed in adenomata of the liver necessitate their extirpation as soon as practicable. A diagnosis without opening the abdominal cavity is, unfortunately, impossible. A solid tumor, small or large, sometimes of the size of a child's head, often movable, which causes subjective difficulties (pain) the existence of which dates back "several years," but which evidently grows, and a diffused enlargement of the liver which in a young or older individual may be marked—this is the usual clinical finding. The recognition is made difficult by the circumstance that the tumor adheres to the liver, is tongue-shaped or even pedunculated, therefore, cannot always be definitely referred to this organ. (Successful operations have been performed by Keen, v. Bergmann, Tricomi, Grube, F. Schmidt and others. Grube's patient died 11 months later from cancer of the liver.)

The history of *carcinomata hepatis* is a comparatively recent one. It is true that literature previous to the nineteenth century contains mention of cancer of the liver (van Swieten, Morgagni, Ruysch, Stoll), but it is not separable from gumma, abscess, cirrhosis, and other hepatic affections. Bayle and Cayol (1812) are usually mentioned as the first who attempted, at least pathologico-anatomically, precise differentiation (from tubercle). The clinical differentiation between primary and secondary carcinoma of the liver is ascribed to Monneret. Prior to the beginning of the sixth

decade of the last century it was believed that carcinoma of the liver was usually of primary development, or, at least, was as often primary as secondary. Bamberger (1864) was the first to state that hepatic cancer was not only rarely a primary affection, but was much more frequently secondary. Virchow proved that the liver is exceptionally fertile soil for the deposition of carcinomata from all parts of the body, and that insignificant primary tumors give rise to enormous secondary neoplasms of the liver; following this, for a brief period, the possibility of primary cancer of the liver was even denied. With increasing knowledge of the histologic structure of the liver and its epitheliomata, the view that carcinoma may actually develop from the liver cell itself has again been expressed, and, unfortunately, is abundantly confirmed by clinical experience. Hanot and Gilbert estimate that for every 8 secondary cancers of the liver there is one primary. Hansemann examined in this respect the reports of the Berlin Pathologic Institute from 1870 to 1889, strictly investigating each individual case, and found among 258 carcinomata of the liver 25 primary neoplasms of the gall-bladder, 2 primary cancers of the large biliary passages, and only 6 primary tumors of the liver (concerning 2 of which he was not positively certain). According to my experience (which is confirmed by my colleague, Eppinger), primary cancer of the liver is comparatively more frequent in Graz than in Prague or Vienna. An insight into the pathogenesis and finer structure of carcinoma of the liver has been given us by the investigations of Naunyn, Waldeyer, Schüppel, Fetzer, Perls, Rindfleisch, Kelsch-Kiener, Weigert, Sabourin, Simmonds, Hanot-Gilbert, Hansemann, and v. Kahlden, which will not be here minutely considered. The clinical differentiation between adenoma and carcinoma, in so far as may be seen from these investigations, is still impossible, a view which has been previously stated.

For a clear conception of the forms of primary cancer of the liver, not only in a pathologico-anatomical but particularly in a clinical respect, it is best to follow the division of Hanot-Gilbert. They differentiate three main types: First, the *massive cancer,* which appears alone; that is, forms but a few nodules; secondly, the *nodular cancer* in which, as is the rule in metastatic carcinoma, the nodules are more numerous; and, thirdly, *cirrhosis carcinomatosa,* which designation is based on the simultaneous occurrence of an epithelial neoplasm and a diffuse cirrhotic hardening of the hepatic tissue. This last-named form included (at least to the greatest extent) the previously described primary epithelial neoplasms called multiple hepatic adenomata (Kelsch-Kiener, Simmonds, Sabourin), and the *diffuse cancerous degeneration* also called *infiltrated hepatic carcinoma* (Schüppel). (Ziegler adds to these a fourth variety of cancer consisting of multiple nodules which develop from the periportal connective tissue and break into the neighboring hepatic tissue.) Finally Orth and Hansemann describe a form which originates in the *small biliary*

passages, and, *tree-like in its course*, spreads through the liver from Glisson's capsule. Strictly speaking, we are, at least, dealing with a neoplasm of the biliary passages, such as primary carcinoma of the hepatic duct, etc.

The very rare *massive cancer* (Gilbert), the carcinoma "*of the nature of a solitary tumor*," develops (preferably in the right lobe) as a solitary, rapidly growing, sharply defined tumor, nearly globular in shape, occasionally of very marked dimensions, sometimes surrounded by intact hepatic tissue (almond cancer) which only late in the course causes metastases in its adjacent areas. The markedly enlarged, diffused organ (sometimes weighing 10 kilograms) retains its shape, and the surface either remains smooth or only small prominences form. The non-implicated hepatic tissue, however, is not cirrhotically indurated; numerous connective tissue branches envelop isolated nodulated portions of the secondary mass. The lymph-glands of the hilus, the gastrohepatic, the peripancreatic, the prevertebral and the mediastinal lymph-glands often show malignant infiltration. Metastases external to the liver are frequently absent; by continuity the cancer occasionally proliferates into the gall-bladder and the right kidney; the peritoneum may also be attacked. Specific emboli may find their way into the hepatic veins, and develop into nodules in the lungs. Perihepatitis and ascites are comparatively rare. The spleen is usually enlarged.

The *nodulated carcinoma* forms numerous secondary masses (occasionally one large and several smaller ones), varying in size from the head of a pin to an orange, and differing in consistence and color. The liver in these cases is often enormously enlarged, its surface becomes nodulated; umbilication is, however, rarer than in metastatic carcinoma with the same dissemination. According to the structure the nodules may resemble the so-called nodular hyperplasia, the trabecular adenoma, and true cancer formation. Distributed connective tissue proliferation in some areas, with induration of the tissue, and atrophy of the parenchyma at other points, as well as perihepatitis, is frequent. Occasionally there is right-sided pleurisy. Enlargement of the spleen is found in about one-half of the cases. The lymph-glands show the same development, likewise the metastases.

In frequency this form corresponds to *cirrhosis carcinomatosa*, in which the nodular (or massive) cancer is not only in juxtaposition with the cirrhosis, but there is a genetic connection between the carcinomatous development and sclerosis of the liver; the latter is sometimes but slight, and, under some circumstances, may naturally also be markedly diffused and the liver enlarged; besides, we find connective tissue proliferation which is uniform, mostly annular, rarely in isolated arrangement; numerous trabecular epitheliomata, that is, adenomata and carcinomata, which are situated deep in the liver tissue (upon section these are quite uniformly distributed, and are often surrounded by connective tissue capsules) or (more frequently) upon the surface of the organ, or, perhaps, protruding

beyond the latter in such a way that cirrhotic granula may be found lying beside cancerous nodules. The volume of the nodules varies; they may be the size of a millet seed or lentil to that of a pea or hazel-nut. This carcinomatous development rarely penetrates into the lymph-glands, but it extends into the venous system much more frequently than any other cancerous variety; in more than one-half of the undoubted cases the secondary mass enters the large afferent and efferent veins of the liver. Secondary nodules are found in the lungs (subpleural); more rarely in the peritoneum (Douglas's sac). Perihepatitis is usually present; ascites is almost invariably added. In more than 50 per cent. of the cases the spleen is enlarged and hardened, dilatation of the subcutaneous veins of the abdomen is frequent, even severe hemorrhages from varicose dilatations of the esophagus occasionally appear (original observation). "Infiltrating cancer," which this form, at first sight, is called at the autopsy, gives the impression of being cirrhosis of the highest grade. Only by microscopic examination do we learn that the islands of cells in isolated areas which resemble resistant hepatic tissue really consist of cancerous masses.

Hemorrhages into and from the carcinomatous liver, which are the result of trauma, of erosion, or of acute congestion and venous hyperemia, are occasionally of clinical importance, especially if the cancerous tissue itself is very rich in vessels, that is, if cavernous capillary dilatations are present. The blood either enters the secondary mass and the neighboring hepatic tissue, or it flows, as I saw in a particularly severe case (potator strenuus), from a portion of the cancer perforating Glisson's capsule into the abdominal cavity. Here it is sacculated by previously formed adhesions in the bursa omentalis, in Douglas's sac, etc.; or it forms a hematoma above the liver (Rollett), or, finally (rapidly fatal), it is freely effused into the abdominal cavity, and is discharged from the peritoneal sac through the inguinal and femoral canals, then infiltrating the subcutaneous tissue.

In regard to "*recovery*" by means of *retrogressive, fatty,* or other metamorphosis which the cancerous nodules of the liver often anatomically present, and the tough, cicatricial masses which are formed thereby, a process which Oppolzer, Bochdalek, and Henoch held to be possible, scarcely anything need to-day be said.

Both sexes appear to be affected by primary cancer of the liver in equal proportions; but cirrhosis carcinomatosa is more frequent in men, presumably on account of its etiologic relation to the abuse of alcohol: this appears as an epiphenomenon to alcoholic cirrhosis. Secondary carcinoma of the liver (for obvious reasons) is more often found in women. The majority of cases occur between the 40th and 45th years; naturally, primary carcinoma of the liver also occurs in advanced age and, exceptionally, in early life, even in children. Heredity and trauma are im-

portant etiologic factors. Malaria is said to be a predisposing cause. Some authors believe there is a signifieant relation to cholelithiasis, since gall-stones are not rarely found besides careinoma of the liver (Biermer).

The statement is commonly made that the clinical history of cancer of the liver presents such a varying picture that a general description is scarcely possible. My own experience leads me to agree with Hanot and Gilbert that, at least in certain stages of the course, the three main anatomical groups may be clinically separated without discussing too-far-reaching theories, although, naturally, the special diagnosis is impossible in some cases. Of massive cancer of the liver, I have collected no individual observations (Gilbert mentions 8 cases with autopsy); but I may cite 7 clinical histories of my own (including autopsies), among them 3 cases of eirrhosis careinomatosa.

CLINICAL TYPES

1. **Massive Cancer.**—Stages of development: Dyspeptie symptoms. Sometimes pain, emaciation, increasing muscular weakness. Pallor. At the height of the disease: Tympanitic abdomen, particularly in the upper areas. Enlargement of the entire liver, and extending below the level of the navel to the anterior superior spine of the ilium. Consistence of the organ markedly firm and hard as wood. Surfaee remains smooth. Final stages: Enlargement (moderate) of the spleen; aseites or dilatation of the veins in the abdominal skin is rare. In some eases: Hypocholia or acholia of the fœces. Jaundice usually absent. Finally, anorexia; rarely vomiting. Constipation (also sometimes diarrhea). Occasionally, and in paroxysms, exaeerbation of the pain. In proportion to the hyponutrition, diminished exeretion of urine. Albuminuria usually absent. Fever (100.4°–102.2° F.) seldom occurs. Course always progressive, rapid, leading to marked cachexia with anasarca. Frequently autotoxic eoma. Total duration: 1 to 5, more rarely, 6 to 7 months.

2. **Nodulated Carcinoma.**—At the onset: Digestive disturbances, aversion to meat, etc., pain in the right hypoehondrium. Exceptionally, early jaundice. After a few weeks, enlargement of the abdomen. At the height of the disease: The enlarged liver, under some circumstances, raises the diaphragm perceptibly, enlarges the right side of the thorax, and causes the hypochondrium and epigastrium to bulge more decidedly. In a downward direction the organ extends below the arch of the ribs, its anterior border below the navel, and at the height of the spine of the ilium it reaches the margo crenatus lienis. The surfaee of the liver is no longer smooth, the edge no longer sharp; these changes are caused by multiple tumors which may be small and numerous, protruding semiglobularly, hard or soft, or may be less in number, voluminous, flat, sometimes even confluent, as if composed of a single tumor. Umbilications are usually

not perceptible. Occasionally a very distinct "shadow" of the liver is seen; sometimes peritoneal friction in the hepatic region. Recognizable continuous growth of the liver under the eyes of the observer. The spleen is either normal in size or only moderately enlarged. In advanced stages of the disease, in more than one-half of the cases, there is serous or hemorrhagic ascites, the amount of fluid often remaining small, and only occasionally necessitating aspiration. Sero-fibrinous effusions occur only in secondary peritoneal carcinosis. The specific gravity of the ascitic fluid varies between 1.010 and 1.020. The amount of albumin may be from 1 to 4 per cent. Pain is almost always present, but varies greatly in degree and character (radiations to the right shoulder, attacks of marked dyspnea, similar to that in diaphragmatic pleurisy). In one-third of the cases jaundice is absent during the entire course; in another third, the icterus is moderate, occasionally only just before death, and, in the last third of the cases, marked permanent jaundice which is not subject to variation develops (by compression of numerous intrahepatic biliary passages, or of the common gall-duct by cancerous hilus ganglia, or by large intumescent gastrohepatic and peripancreatic lymph-glands). Constipation. During the latter period there is vomiting, especially when there is rapid growth of the tumor. Acholia in consequence of hepatargy is rare. Profound anemia, cardiac asthenia, variable temperature, usually normal, but toward the end with moderate fever, may occur. Long-continued high fever is observed only in isolated cases when the tumor enlarges very rapidly; sometimes it may be combined with chills, it may be remittent, or even continuous. Total duration: A month to a year; the average is 3 to 6 months. The younger the individual, the more rapid, as a rule, the course. Death may take place in coma; more frequently it occurs with the symptoms of sheer exhaustion. Besides the ordinary form just depicted, Hanot and Gilbert describe quite a number of special "varieties." I concur with them only so far as their observations accord with my own experience and that of other authors, and must reiterate that the clinical symptoms of nodular cancer are, in fact, but little less characteristic and uniform than those of other types of carcinoma hepatis. Fever is occasionally prominent in the clinical picture, or the cancer may betray itself by the predominance of a cachexia, while, in other cases, the body retains its fulness for some time. Carcinoma may, during long periods of its course, produce no other functional symptoms than paroxysms of intense pain: in other cases, continuous vomiting is a prominent feature. In conclusion, permanent jaundice may play a leading rôle.

3. **Cirrhosis Carcinomatosa.**—Symptoms at the onset: Dyspeptic disturbances (loss of appetite, sense of weight in the stomach, flatulence, eructations, vomiting). Indefinite pain, loss of strength, emaciation, anemia. Frequent epistaxis. Early enlargement of the abdomen. At the height of the disease: Cachexia, ascites, often increasing so rapidly that

aspiration becomes necessary. Dilated veins of the abdominal skin. The liver may be enlarged, and increase under the eyes of the observer; or, it cannot be palpated, and decreases in size. If still palpable, the surface appears coarse, nodular, and occasionally granular. The spleen is often enlarged (but only moderately), even palpable; sometimes, however, it is normal. Jaundice is found in all the well-marked cases; it occurs early or sometimes even late. There may be constipation, or, on the other hand, diarrhea; toward the end the feces become acholic. Hematemesis and melena appear. Fever plays no rôle; toward the end there is edema. The total duration of the disease is 1 to 16, upon an average, 4 to 5, months. After anasarca has appeared death occurs in coma and from extreme exhaustion.

As will be noted, in the *development* of the three anatomical types of primary cancer of the liver, the same constitutional disturbance, disorders of digestion, as well as very similar, but only slightly characteristic, pain, and the wretched appearance of the patient are common. Absence of jaundice is the rule. Massive cancer, and cirrhosis carcinomatosa with a relatively small liver, often exist for some time without causing local signs. Symptoms such as those of Bogdan (flaky, wine-red discoloration of the cheeks, and varicosities of the superficial capillaries of the same region occurring in gastric and uterine carcinomata), or that of Mettenheimer (marked emaciation of the epigastric region with an especial distortion of the navel, "navel-fold," in cancer of the abdomen) is scarcely of decisive import. The tenacity and, still more, the visibly rapid progression of the previously mentioned symptoms would incline us to the first suspicion rather than to the view of a mild gastric or intestinal affection. But a careful study of the clinical reports of other authors and my own experience teach me to beware of the danger that lurks in the latency of the disease and the absence of objective symptoms referable to the diseased organ, which, as a rule, continue until it is too late for the physician to accomplish anything effectual. Of the value in such cases of a possible diagnostic exploratory laparotomy we shall speak later. In the transitional period, the main stress is to be laid upon the parallelism of an increasingly rapid loss in weight, and a rapid enlargement of the entire liver. Quickly as a carcinomatous liver grows, as a rule only certain forms of congestion develop, and these are usually not difficult to exclude. There is scarcely a doubt of the fact that when a palpable tumor of the liver can be determined, it is too late for effectual operative interference. Two initial symptoms upon which much stress is often laid are hyperleukocytosis and the decreased amount of urea in the urine; the first is of minor importance, and the second of scarcely any differentio-diagnostic value. In my experience, leukocytosis (and especially certain forms of digestive leukocytosis) favors cancer; I have reason, however, to doubt that this actually occurs early. The symptom, too, is variable. In my

cases, the number of leukocytes to the cubic millimeter varies between 6,500–18,000; but my experience compels me to differ from the view that urea and chlorin are invariably decreased in the urine in the early stages of cancer of the liver. The amount of urine and nitrogen in carcinoma hepatis depends, in the main, upon the degree of inanition existing, and a rapidly increasing ascites may influence the excretion of water. In the earlier stages, and even at the height of the disease, I found medium amounts of urea, and it was possible even to bring some patients temporarily into N-equilibrium! Alimentary glycosuria plays no diagnostic rôle, and acholia is not an actual early symptom.

DIAGNOSIS

At the height of the disease we may adhere, in a diagnostic respect, to the previously described types. In practice, as soon as a well-developed cachexia is observed, or a palpable tumor of the liver appears combined with symptoms of compression in the region of the portal vein and in the biliary passages, a special differentiation is only of importance as an indication that operation is unnecessary. All patients succumb soon or late, and, therefore, suffer to about the same extent. Ascites is a frequent hindrance to the recognition of the affection in its advanced stages. Special diagnostic consideration of compression (simultaneous) of the biliary passages and the portal veins has already been referred to; all carcinomatous tumors of the liver are characterized by the great increase in volume which is not significant in any other affection of the liver. The cases of soft cancer of the liver, in which the sensation through the abdominal wall simulates fluctuation, are rare exceptions. In a differentio-diagnostic respect, besides massive cancer, we must consider the following: Melanomata (which usually also cause enlargement of the liver with a smooth surface, but in most cases are secondary to neoplasms of the eye or skin, and give rise to melanuria), Hanot's hypertrophic cirrhosis (having a slower course without pain, cachexia and marked anemia, hypercholia, that is, so-called poussées, jaundice, hard splenic tumor), Hutinel-Sabourin's cirrhosis adiposa hypertrophica, certain forms of congestion and enlargement of the liver in diabetes, malaria, syphilis, leukemia, pseudo-leukemia, amyloidosis and cholelithiasis. Many forms of nodular cancer so completely fill the abdominal cavity that it becomes difficult to determine the point of origin. Perplexity in diagnosis may also arise when the cancerous nodules coalesce and form a few larger structures or a tumor. Confusion with secondary hepatic cancer is a frequent occurrence. When a tumor of the liver is present we should never fail carefully to examine the entire body for a primary neoplasm (by palpation of the stomach, digital examination of the rectum, of the vagina, etc.). In opposition to Lenbe's view, I must state that the determination of the acidity of the digesting

stomach permits no decisive conclusion; even in unquestioned primary cancer of the liver, free hydrochloric acid may be absent. Pulmonary and pleural cancers, as previously indicated, occur secondarily somewhat more frequently in the course of cancer of the liver; similarly mediastinal and peritoneal tumors, but rarely growths in the abdominal wall (umbilicus) and small tumors. The nodules of primary carcinoma are generally harder than those of secondary cancer. The umbilication is frequently unrecognizable. In secondary cancer of the liver there may be no enlargement of the organ, and the tumor of primary cancer of the gall-bladder may, in most cases, be diagnosticated with certainty according to criteria which have been enumerated. Exceptionally, carcinoma ventriculi causes distributed tumor formation which resembles liver nodules, and certain neoplasms of the omentum may simulate the form of the liver. Cancer of the right kidney has been repeatedly confounded with carcinoma of the liver. The nodular surface of nodular cancer is common also to syphilitic liver, to multilocular echinococcus, and to liver cysts of non-parasitic origin. We differentiate by the slighter resistance of the protuberances, the slow development and the late cachexia, besides the history and other symptoms (for example, the condition of the spleen). Syphilomata may occasionally appear as isolated tumors of the size of a fist (more or less painful) below the right arch of the ribs in the diffusely enlarged liver, which is of normal consistency. Such gummata have been mistaken for cancer and extirpated. But, inversely, syphilomata have been assumed, and mercury has been administered until, on exploratory laparotomy being later performed, an inoperable soft cancer has been revealed. Even confusion with chronic abscess of the liver has occurred. In cirrhosis carcinomatosa the symptoms vary according to the degree of cirrhosis present, this depending upon the situation, the number and the size of the cancer nodules, that is, the infiltrated carcinoma mass. Correspondingly the liver is sometimes large, or very large, and confusion with nodulated cancer or secondary hepatic carcinoma is possible; under these circumstances marked ascites is a diagnostic warning. In another series of cases with but slightly enlarged liver which slowly decreases under observation, especially if alcohol may be regarded as the cause, we are inclined to make a diagnosis of cirrhosis. As is well known, there are also forms of the latter disease which run a subacute course, in which marked parenchymatous degeneration of the cellular elements of the liver plays an important rôle. Generally, however, it is cirrhosis carcinomatosa in which the rapid development and severe nutritive disturbances are peculiar, and which soon terminates in death. Finally, there are cases in which the right-sided pleural exudate early forms and appears to explain the entire clinical picture.

In extremely rare cases only does the enlargement of superficial lymph-glands assist us in the diagnosis of cancer of the liver. The jugular gland,

especially, plays no rôle. Enlargement of the inguinal glands is somewhat more frequent.

Except from the stroma, *secondary cancer of the liver* originates exclusively from cellular elements which have been detached from an extrahepatic carcinomatous neoplasm, and by the lymphatic, arterial, and venous channels have reached the liver to which they adhere, then proliferate in the capillaries. There is scarcely an epithelial tissue from which the liver may not thus be secondarily affected. Obviously a special tendency to infection of the liver is found in carcinomata of those abdominal organs which are closely related to the portal vein (stomach, rectum, intestine, pancreas, biliary passages). I am in possession of accurate histories and autopsy reports of 27 cases of secondary cancer of the liver. The primary neoplasm was 15 times seated in the stomach (in 2 of these cases only the left lobe showed circumscribed implication), 4 times in the gall-bladder, in 4 cases in the rectum, in 2 in the kidney, and in 2 in the esophagus. Hepatic metastases are, however, also found in primary cancer of the testicles, of the seminal vesicles, of the prostate gland, of the uterus, of the ovary, of the breasts; in the lungs they are exceedingly rare. The neoplasms may occur secondarily outside of the actual portal vein area (due to other metastases), having reached the portal vein system prior to their deposition in the liver. The secondary cancer of the liver possesses an extraordinary tendency to form multiple nodules, which, in regard to their number, volume, consistence and shape, do not differ from cancer nodularis. As a rule, the liver shows very diffuse enlargement, attaining a weight of from 5 to 10 kilograms); its surface becomes nodular. Exceptionally the secondary growth is limited to the left lobe of the organ; more rarely does diffuse infiltration occur (Litten). The conspicuous tendency to retrogressive metamorphosis, in contrast to primary carcinoma, is usually looked upon as the cause of the marked umbilication. Breaking down of the cancer may also lead to the formation of cysts, to hemorrhage, even to rupture and bleeding into the peritoneal cavity.

TREATMENT

The limits of operabilitas hepatis quoad resectionem, as it is called by Langenbuch, as well as the results of experimental investigations and of operations already performed in man (and even excluding from consideration the great danger of hemorrhage), are such that the justification for, and the technical possibility of, the removal of malignant tumors of the liver is out of the question. The animal experiments of Ponfick, which have become celebrated, and later those of Meister, Kusnezow, Penski and others, are decisive indications for this proceeding in surgery of the liver, in that they have absolutely proven that the removal of even a relatively large portion of the liver may be borne without serious damage.

Any one who has carefully examined a number of carcinomata and sarcomata of the liver must admit that the reason why tumors of the liver are rarely removed is not the impossibility of the operation, but the difficulty of early recognizing the indication for the operation, and its consequent poor results explain why surgery, which is constantly advancing into wider domains, unfortunately cannot completely master this organ. Occasionally it may occur that a gastric, intestinal or gall-bladder cancer may be removed, also simultaneously a portion of the liver, which is exclusively the seat of a beginning secondary carcinomatous development. In all other cases, metastatic carcinoma or sarcoma will only be an absolute contraindication to operative procedure. At most in a very small fraction of the rare primary malignant neoplasms of the liver is there any indication for surgical treatment. Certain types of carcinoma, for example cirrhosis carcinomatosa, which is observed with comparative frequency, and also most cases of nodular cancer, are not suitable for this kind of treatment. The peculiar structure of the liver which greatly favors the rapid distribution of an originally circumscribed affection to wide areas of the organs, the multiplicity of the malignant neoplasms which soon become obvious, the difficulties of an early diagnosis which have been previously described, the history which for the most part only points to a disease of the intestinal system, are all observed too late to be absolute indications for operative treatment directed to the liver. Many surgeons, therefore, hold that these early symptoms above described, which with more or less positiveness point to a serious abdominal disease, indicate an exploratory laparotomy. In this manner a tumor of moderate size, from that of a hen's egg to an apple, and favorably situated, might be removed; personally I know of nothing that contraindicates an exploratory laparotomy. If we consider, however, how often on such vague signs this operation would be resorted to in practice, and how frequently, from obvious diagnostic errors, it would be practised unnecessarily, it is easy to understand that the carrying out of such a proposition would meet with objections on the part of the patient and his relatives and would even be objected to by those physicians who themselves have committed a few errors. Therefore, many favorable circumstances must be combined to enable us to perform resection of the liver to the benefit of the affected patients. To Hochenegg belongs the honor of being the first successfully to remove a cancer of the liver from its point of origin in the gall-bladder. A case of Lücke's is very instructive as a guide to the decision for the operation. This case was published in 1891, and, on account of the successful removal of the left lobe of the liver, it was generally looked upon as an example of a pure primary cancer of the liver until a more recent publication of Madelung (1898) showed that it was only a syphiloma.

ECHINOCOCCUS OF THE LIVER[1]

By E. STADELMANN, Berlin

Clinically and anatomically we may differentiate two varieties of echinococcus in man: 1. Cystic echinococcus; 2. Multilocular or alveolar echinococcus. We know that the echinococcus cysticus may develop (Siebold, Küchenmeister, Leuckart) whenever the ova of the tenia echinococcus which lives in the intestinal canal of the dog, the wolf, the jackal, etc., are found in the gastrointestinal canal of man. The embryo, which is set free by the digestion of its chitin covering, burrows through the intestinal wall, reaches a branch of the portal vein, and enters the liver with the circulation; here it lodges and is transformed into an echinococcus cyst. Naturally, the echinococcus develops not only in the liver but also in many other organs (lungs, spleen, brain, kidneys, etc.), provided it can find ingress by means of the circulation or the lymph channels. In its early existence the echinococcus cyst develops but slowly, first to the size of a tubercle, later to that of a pea, but it is a firm nodule with a thick wall which is composed internally of muscle and vessels (parenchymatous layer), and externally of a tough, lamellar layer of tissue (cuticula). In the affected organ, and around the cyst which is filled with a clear, non-albuminous fluid, a tough connective tissue capsule forms which may be easily peeled from the nodule, and in which are many vessels by means of which the echinococcus is nourished. After four to six months the growing vessel attains the size of a walnut, and in its interior (endogenous), very rarely externally (exogenous), daughter-cysts now begin to develop (secondary hydatid cysts), even granddaughter-cysts (tertiary hydatid cysts) which are partly sterile, and partly contain the heads (scolices) of the echinococci. These heads have the characteristic shape, and are supplied with suctorial discs and a crown of hooklets. Usually the daughter-cysts become detached from the inner parenchymatous wall, and float free in the fluid, so that in such a mother-cyst hundreds of daughter-cysts may be found.

The development of the multilocular echinococcus is different. Virchow first recognized this as a parasite; it forms a coarse tumor composed of

[1] See also the article "Echinococcus Disease" by Prof. E. Peiper in the volume on "Diseases of Metabolism and of the Blood; Animal Parasites; Toxicology," page 526.

vesieles the size of a pea. In contrast with the cystic echinococcus, its growth, therefore, takes place by exogenous proliferation in that one echinoeoecus vesicle attaches itself to another. It betrays the same nature as malignant neoplasms, and not only attaeks the parenchyma but also the blood-vessels, such as the portal vein, the hepatic veins, the hepatic arteries and the lymph-vessels. Constriction may take plaee, and, as in malignant neoplasms, give rise to metastases. It is a mooted question whether multiloeular eehinococeus is identieal with cystie echinococcus, or whether we are dealing with two different varieties of teniæ. A number of faetors favor the latter view; for instance, the circumstance that eehinoeoccus multilocularis occurs in definite localities (South Germany, Austria, and parts of Russia) while in other regions (Iceland, Meeklenburg, and Pomerania) the echinococcus cysticus preponderates, and also that some authors by feeding experiments with the echinocoeeus multilocularis suceeeded in producing this parasite experimentally; this statement, however, is denied by other authorities. Finally, the seolices of these forms are said to differ from eaeh other, particularly in the shape of their hooklets. No matter how this may be, the question whether we are dealing with two different varieties of eehinocoeei or whether the same echinococcus produces two different forms of the disease, must be looked upon as still in dispute.

A. ECHINOCOCCUS CYSTICUS

PATHOLOGICAL ANATOMY

The origin and development of the vesicles, as well as the eonfiguration of the walls of the cyst, have already been deseribed. Besides daughter-eysts, the vesicles contain a elear, almost invariably non-albuminous fluid with a specifie gravity of from 1.009 to 1.015, and varieties of sugar (grape-sugar, inosite), succinic acid and salts (principally sodium chlorid). According to the investigations of different authors (Mourson and Sehlagdenhauffen, Brieger), toxic substanees are also present whieh may be assumed to be ptomains, since, when the contents of the eyst are injeeted into animals, they produce collapse and death. Echinoeoeei are usually found in the right lobe of the liver. The vesicles may develop in any region of the liver, upon the upper surface (the eonvex), upon the lower surface (the coneave), centrally as well as subphrenically. The symptoms of displacement of other organs, as well as the extent of the growth, frequently also depend upon the seat of the echinococeus, and this again produees the clinical symptoms as well as the diagnostic determination of the pathologic proeess. We would digress too far if we entered upon the individual points which are self-evident. It need only be stated that the tumor may extend to the erest of the ilium, may force the diaphragm up toward the pleural cavity, may sometimes be found deep in the peri-

tonal cavity, often causing the abdominal walls to assume a globular shape, at other times it is prominent upon the surface of the liver. The death of the echinococcus may result from various causes (entrance of bile into the echinococcus sac, nutritive disturbances in consequence of pressure from other organs, etc.), then the contents of the sac become turbid, milky, and resemble pus, but are without pus cells; the fluid becomes more and more inspissated, the friable mass consists of cholesterin, calcium carbonate, phosphate, and the remains of scolices, but particularly of the very resistant hooklets.

True suppuration of the echinococcus cyst is not rare. This develops in consequence of trauma or from the entrance of pathogenic agents (bacteria) into the interior of the echinococcus sac. It is facilitated when an avenue of communication forms between the cyst and the bile channels so that bile and also bacteria penetrate to the cyst. This not only causes the death of the echinococcus but suppuration of the contents of the cyst, after which all the symptoms of hepatic abscess appear.

SYMPTOMATOLOGY

The echinococcus produces no symptoms in the initial stages. Quite large cysts may remain latent, and be discovered only accidentally at the autopsy. This is due to the slow growth of the cyst, as well as to the slight effect it produces upon the surrounding tissue. Examination will reveal palpable changes only when the tumor is quite large and situated upon the surface. Even such a tumor may readily be confounded with others since it is rarely characteristic. A cyst can be diagnosticated only when it is soft and shows fluctuation, and thus arouses a suspicion of echinococcus. A peculiar vibration in the cyst upon percussion and also upon palpation has been much spoken of, and to this the name "hydatid thrill" has been attached. This is said to be produced by the collision of the daughter-cysts in the sac, but it may be doubted whether this explanation is correct. The hydatid thrill has also been detected in sterile cysts. The recognition of tension of the membrane is also important, for with decided tension this thrill does not appear. The hydatid thrill is best developed when several fingers of one hand are placed upon the cyst and the fingers of the other hand tap successively, as if performing ballottement. The hepatic area in which the cyst is located influences greatly the production of the different symptoms. Cysts which form upon the lower surface of the liver extend downward into the abdomen; they may produce compression of the porta hepatis, of the common bile duct, of the portal vein, of the veins of the intestines, etc., with their corresponding sequelæ. Naturally, the entire right lobe of the liver is enlarged, while the greater portion of the cyst is insusceptible to examination. Echinococci which develop upon the upper portions of the liver frequently force the diaphragm

upward and decrease the thoracic space (subphrenic seat), so that the differential diagnosis from pleural exudate becomes necessary; the thorax is distended upon the right side, the lung more or less compressed; this gives rise to cough, dyspnea, and, by inhibiting cardiac activity, also circulatory disturbances and displacement of the heart to the left. An echinococcus of the left lobe of the liver, which is rare, causes symptoms of displacement of the heart, of the stomach, and finally also of the left lung, and produces not only marked enlargement of the left lobe of the liver but even an entire change in the organ's position. Central echinococci give rise to general hepatic enlargement less often than to change in the form of the liver.

A large echinococcus cyst of course produces numerous symptoms due to enlargement of the liver, and combined with this also symptoms of displacement which are, however, not typical. Fulness, signs of pressure in the abdomen, dyspnea, oppression after the ingestion of food are the usual complaints, as in any other tumor of the liver.

Suppuration of the echinococcus cyst produces new symptoms, such as remittent or intermittent fever, chills, pain, especially in the hepatic region, which radiates to the right shoulder, great constitutional disturbance, signs of local peritonitis, of perihepatitis, finally also of general pyemia. None of these manifestations can be differentiated from those of abscess of the liver.

Rupture of the echinococcus cysts and the penetration of the parasite into other organs call for a special description. Aside from tumors this is mainly due to a too voluminous growth of the cyst, and especially to suppuration in the same. When an echinococcus ruptures into the biliary system the parasite is killed, and, in consequence of secondary infection on the part of the bile which enters, the contents of the cyst frequently suppurate. The fluid contents of the cyst may be discharged into the intestine through the small, communicating biliary channels while the cysts themselves remain. If rupture occurs into the hepatic or common ducts it is possible that the hydatid may be discharged, and it may enter the intestine, perhaps with accompanying symptoms of an attack of cholelithiasis; synchronous with this the cyst rapidly collapses.

If the hydatids remain in the common bile duct, stasis jaundice may be the consequence, also the symptoms of acholia, etc. By admixture with bile, the contents of the cyst show marked icteroid discoloration.

Perforation into the stomach occurs when the cyst is situated in the left lobe of the liver or upon its lower surface. Adhesions to the wall of the stomach must have preceded. When the rupture occurs, the patient vomits profuse watery masses containing hydatids, and the cyst collapses. A portion of the contents naturally finds its way into the intestine.

Perforation into the intestine is brought about in the same way by local peritoneal inflammation and adhesions which run their course ac-

companied by pain. After rupture, the cyst rapidly collapses, the feces become watery, and sometimes contain blood, hydatids, or their remains. The lesion of communication may close or it may persist. In the latter case fecal contents find their way into the liver, and there develop a fecal abscess.

Perforation into the peritoneal cavity results when there have been no adhesions to neighboring organs, as is so frequently the case, but contusions, trauma, sudden violent exertion, force or pressure, etc., may be the cause. The results of rupture are severe shock, syncope, great abdominal pain, mental disquietude, the sense of impending death, or even sudden dissolution. The patients feel as though a part of the abdomen had been torn; they are pale, anxious, and distressed. Urticaria frequently appears in consequence of the toxins which have developed in the fluid. If the patients do not succumb to the rupture the hydatids may still further develop in the abdominal cavity, and if the generators of infection contained in the fluid of the cyst are expelled into the belly, peritonitis follows. This process is much more favorable if adhesions to the peritoneum have previously formed, and if the cyst ruptures into an encapsulated portion of the abdominal cavity, so that diffuse peritonitis does not result.

Rupture into the urinary passages is very rare, but may lead to renal colic and hydronephrosis when the hydatids are caught in the ureter. When they are voided by the urinary channels, we find in the urine cysticerci vesicles which are occasionally bile-stained.

Rupture into the pleural cavity results in cysts in the subphrenic space. According to the composition of the exudate, we then have the signs of serous or purulent pleurisy. By exploratory puncture we obtain some of this fluid, and, microscopically, we are sometimes able to make a correct diagnosis by demonstrating hooklets or portions of the membrane. The slight amount of albumin, the clearness of the serous fluid, or its bile-stained appearance, may awaken our suspicions that it comes from an echinococcus cyst. The rupture of the sac gives the patient a sensation as though something within the chest had been torn, then follow a feeling of anxiety, dyspnea, or even collapse. The pulse becomes small and frequent, and with these symptoms death may soon occur. If the contents of the cyst are purulent, the well-known symptoms of empyema with pain and fever are produced. The empyema, if not recognized in time and treated by operation, may rupture externally or into the lung. In the latter case, occasionally with severe paroxysms of coughing, great numbers of hydatid vesicles are coughed up, and complete discharge of the contents of the cyst as well as cure results. In other cases a pulmonary abscess forms; pneumothorax from the rupture of an exudate into a bronchus, and even direct rupture of the contents of the cyst into the lung, without the development of an empyema such as has been described or of pleurisy, have been reported. If rupture into the bronchi or the trachea

occurs too rapidly in consequence of the great mass of fluid and hydatid cysts expelled, the patient may be suffocated. Large cavities may develop and produce corresponding phenomena. Upon rupture of an echinococcus cyst into the lung the sputum is frequently bile-stained.

Rupture into the pericardium is most infrequent, and probably in all cases soon causes death by paralysis of the heart.

Rupture into the portal vein produces multiple abscesses in its various branches.

Rupture into the vena cava permits the entrance of hydatids into the circulation, thence to find their way into the right heart, and later into the pulmonary veins, producing pulmonary emboli with all the symptoms of a hemorrhagic infarct or even a pulmonary abscess.

Suppurating cysts may also rupture externally through the abdominal wall, the same as hepatic abscesses, unless they are early recognized.

DIAGNOSIS AND TREATMENT

Small echinococcus cysts cannot be recognized. They are, however, occasional autopsy findings. The recognition of large cysts is mainly due to their position, but even then they are frequently overlooked, especially if they produce no symptoms and their position is concealed. If we discover a tumor which we suspect to be an echinococcus, this diagnosis rather than that of other tumors, particularly malignant ones, is favored by the slighter pain, the round, smooth surface, the mild disturbance of the general condition, the slow growth, the tense, elastic consistence, perhaps also by a positive hydatid thrill. If remittent or intermittent fever coexist, the differential diagnosis between abscess of the liver and a suppurating echinococcus cyst may be impossible without surgical intervention.

In the diagnosis of a subphrenic cyst the X-rays, as well as the observation of Litten's diaphragm phenomenon, will be of value. In a pleural exudate the diaphragm is forced down, in a subphrenic echinococcus, on the other hand, it is forced upward, while the remaining clinical signs in these pathologic conditions differ but little from each other.

Cysts of the spleen, situated near the left lobe of the liver, may be mistaken for echinococci, also those of the pancreas and even of the ovary, as well as dropsy of the gall-bladder and a right-sided hydronephrosis.

In many cases exploratory puncture with the subsequent chemical and microscopical investigation of the cyst contents which have been obtained may clear up and decide this question. This is, therefore, an important diagnostic aid which may be employed without danger in case of a puncture of the pleural cavity, provided antiseptic precautions are taken. On the other hand, puncture of the abdomen is decidedly hazardous. Even thin cannulæ will produce holes in the tense cyst wall from which other contents may readily ooze, and, particularly if they are infectious, lead to

general peritonitis. Therefore I earnestly advise that puncture be performed only upon the operating table, and when operation is to follow immediately; in the interests of the patient it is well to leave a definite diagnosis to the surgeon. It is certainly fortunate for the patient if the physician decline the triumph of making the decision, and work hand in hand with the surgeon. He may do this all the more conscientiously since, if echinococcus be present, the only possible treatment is by operation. The cyst must be opened, and, if it prove to be an echinococcus cyst, the contents must be evacuated. How this is to be done, and which method of procedure is the best, is difficult to decide, as the situation of the cyst in the individual cases may necessitate different methods. Neither can it here be positively stated whether the single or the double method will give better results. At all events, it should be the earnest endeavor of every surgeon to avoid in every possible way the infection of the neighboring organs by the fluid evacuated from the cyst. After the parasite has died some cysts heal by calcification.

Drug treatment is wholly ineffectual, and it is advisable not to attempt it.

B. ECHINOCOCCUS MULTILOCULARIS SEU ALVEOLARIS[1]

The question as to the manner in which multilocular echinococcus differs from cystic echinococcus has been discussed, as well as the nature of its development. The typical difference is that multilocular echinococcus forms in the cavities of the liver (bile ducts, blood-vessels, lymph-channels) and extends by gemmation, destruction going on in the interior of the colony, and thus cavities differing in size are formed, also that its action in the liver resembles that of malignant neoplasms since it destroys the actual hepatic tissue and at the same time causes an extensive connective tissue proliferation. In consequence of this mode of development, the appearance of the liver in echinococcus multilocularis may vary greatly.

If the tumor is situated in the interior of the organ, the form of the liver is not necessarily altered and the disease process is revealed only by section. In other cases, nodular protuberances of varying size are found upon the surface, and between these are connective tissue retractions; occasionally distinct echinococcus cysts are recognizable in the nodules. The consistence of the liver is sometimes very dense owing to a large deposit of chalk frequently found in the connective tissue. In old echinococcus cavities in the interior this condition is found as the consequence

[1] For the literature compare the following works: *Hoppe-Seyler* in "Die Krankheiten der Leber von Quincke und Hoppe-Seyler"; *Nothnagel's specielle Pathologie und Therapie*, XVIII, Theil 1; *Vierordt, Berliner Klinik*, Heft 28, and the monograph upon "Multilocular Echinococcus" published in Freiburg in 1886; *Posselt, Deutsches Archiv für klin. Medicin*, LIX.

of decomposition which has taken place. The liver is usually, and sometimes greatly, enlarged, cases of echinococcus multilocularis having been described in which two liters of fluid were evacuated from the cavities. Vicarious hypertrophy of the liver substance, chiefly of the left lobe (the echinococcus usually develops in the right lobe), has been reported.

The small tumors are white to yellowish green in color, with a distinct line of demarcation from the surrounding, dark, hepatic tissue.

Section of the tissue produces a grating sound, the cut surfaces reveal numerous apertures (alveoli), hence the tissue resembles a honeycomb, and the echinococcus has been designated "alveolar." These alveoli are of different sizes, ranging from that of a pin's head to that of a pea, a walnut, an apple, a child's head, etc., and between them the connective tissue is increased. As these cavities coalesce a peculiar picture may develop; the consistence of the contents varies from that of a gelatinous material to a tough, dry, friable, even calcareous mass which is usually a bile-stained yellow. In consequence of the great destruction going on, enormous cavities may occasionally form; these are partly filled with fluid, partly with compact masses permeated by connective tissue striæ which represent the remains of large blood-vessels and bile channels. The contents of the cavities often have a thready, cream-like, gelatinous, more or less bilious or even brownish appearance, and, according to microscopical and chemical investigation, they are composed of mucus, calcium, cholesterin, remains of blood, bilirubin, hematoidin, remains of echinococci, hepatic tissue, albumin, biliary coloring matter, bile acids and fat.

Macroscopically and microscopically the hepatic tissue is greatly altered. The bile ducts are sometimes dilated by the tumors growing within them, at other times are compressed by these growths. The common bile duct may be occluded from compression; the same is true of the cystic and hepatic ducts. This produces various other anomalies such as absolute occlusion of bile with severe jaundice, dropsy of the gall-bladder, etc.

In consequence of compression of the portal vein, stasis arises with enlargement of the spleen, ascites, etc. The lymph tracts are also frequently implicated by tumors, and consequently have a peculiar appearance; they are filled with echinococci and resemble a pearl necklace or a rosary. This latter condition is even observed in the lymph-glands at the porta hepatis. Metastases of echinococci are also found in the lungs, the spleen, the mediastinal glands, the pericardium, the peritoneum, the pleura, and even in the endocardium. Besides the already described contents of the individual cavities the microscope reveals that, surrounding these tissues, a structureless membrane is first formed which belongs to the echinococcus itself and is usually distinctly layered, and subsequently an extensive connective tissue proliferation which may be regarded as a reaction of the liver to the foreign body that has encroached upon its substance. Upon the inner surface of the cavity there is also a parenchymatous layer. Hooklets and

scolices are frequently absent from the primary tumor, and this coincides with the existing tendency to death and destruction of the parasite; they are, however, present in recent growths and in metastases. The membrane of the parasite itself, which corresponds to the cuticula of the cystic echinococcus, naturally consists largely of chitin.

SYMPTOMS AND DIAGNOSIS

Alveolar echinococcus runs its course for a long time without symptoms, then pressure signs, a sensation of tension, pains in the epigastrium, loss of appetite, nausea, etc., appear—symptoms which are by no means typical. Fever and even jaundice (pressure symptom) may be present. The patient's sufferings increase, and he begins to emaciate. Objectively we find a more or less enlarged liver of increased consistence, and if the echinococcus tumor be susceptible to palpation hard, nodular masses are felt. Occasionally we obtain distinct fluctuation even with a very large, superficially situated, decomposing echinococcus cyst. In other cases in which there is a hard, tough tumor of the liver, hypertrophic hepatic cirrhosis is simulated.

Sometimes the symptoms of jaundice due to compression of the common bile duct may be particularly prominent and combined with all the familiar manifestations of icterus gravis. In other cases, the signs are those of portal vein stasis with ascites, splenic tumor, etc., in consequence of the compression and constriction of this vessel which dominates the clinical picture, as well as the familiar gastrointestinal symptoms which are so frequent in portal vein stasis, such as constipation, diarrhea, loss of appetite, etc. Death results from increasing emaciation and exhaustion, edema, and cachexia, usually after a very long duration of the disease.

The *diagnosis* can rarely be made with anything approaching positiveness. In the differential diagnosis hypertrophic hepatic cirrhosis and cancer of the liver will usually come into question, according to the symptoms of the individual case. Age, the environment of the patient, the examination of the gastric juice, fever, and enlargement of the spleen will usually protect us from error in the differentiation from carcinoma. When we suspect the existence of a cavity in the liver, exploratory puncture and the subsequent examination of the contents of the cyst thus obtained will lead us to a definite opinion. Of course, in echinococcus alveolaris the nature of the contents of the cavity varies according to its seat. Sometimes its constituents are biliary, such as biliary coloring matter, bile acids and cholesterin; at other times fatty, if communication of the tumor with the lymph tracts has occurred; and in still others, upon communication with the blood-vessels, blood may be present.

In contrast with the fluid contained in the cavities of cystic echinococcus, the echinococcus alveolaris contains extremely large quantities of albumin.

When we suspect echinoeoeeus alveolaris, exploratory laparotomy and the examination of an exeised portion of the tumor is elearly indieated.

Amyloid liver and hepatie abscess, as well as eystie eehinoeoeeus, may all be considered in the differential diagnosis. The first two are easily excluded. In the last, the result of the examination of the fluid obtained by exploratory puneture is decisive.

PROGNOSIS AND TREATMENT

The disease is nearly always fatal. We may, however, hope that with inereasing knowledge of the malady and a timely diagnosis, proper treatment, partieularly early operative measures, may be instituted. These should eonsist of extirpation of the tumor and resection of the affeeted portion of the liver, but they ean only lead to favorable results when the operation is performed in the initial stages of the affeetion. The possibility of this is demonstrated by faets, sinee a eure of the disease in this way has eertainly been effeeted (Bruns[1]).

[1] *Bruns' Beiträge zur klin. Chirurgie,* XVII.

GALL-STONES

By E. NEUSSER, Vienna

Cholelithiasis is a border-land for the physician and surgeon. Both are familiar with its symptom-complex, and must work hand in hand to discover the etiology, to make the diagnosis and prognosis certain, and to formulate its therapeutic indications.

Many results in this realm we owe to the physician, many to the surgeon, who actually makes autopsies *in vivo*. But, in spite of numerous researches and statistics from both sides, they have not completely elucidated even the most vital questions in cholelithiasis, such as the question of jaundice, nor even that of the mechanism of gall-stone colic.

SYMPTOMATOLOGY

The etiology of gall-stone formation, the important factor which to both would be explanatory, and would also decide the treatment, is still obscure: here aseptic inflammation, there bacterial infection; upon the one side confidence in sterile bile, upon the other the specter of the bacterium coli, the exaggerated fear of all possible anatomical complications, and the old belief in the efficacy of a mineral spring cure.

It cannot be denied that, at the present time, the surgeon's word carries the weight of authority, and already almost identifies an early operation with a radical cure. Yet he may, any day, be surprised by the discovery of an anti-bacterial serum which will render *gall-stones inert*, and the operation for radical extirpation will meet the same fate as did tracheotomy in true diphtheria.

Until this fortunate epoch arrive, the clinician, in justice to both parties, must limit himself in the given case to protecting the patient from the two extremes, eagerness to operate and failure to recognize the right time for operation.

The usual symptoms of cholelithiasis are pain in the right hypochondrium which radiates to the back and the region of the shoulder, which either sets in suddenly or rapidly increases in severity, also eructations and vomiting, chilliness or well-developed chills, sensitiveness to pressure in the region of the gall-bladder and succeeding jaundice. In the overwhelming majority of cases these symptoms will justify a diagnosis of cholelithiasis; nevertheless, there are also cases in which the same symp-

tom-complex, in the same sequence, is present, and to our surprise, instead of finding the expected gall-stones in the feces, the passage of echinococcus cysts completely changes our diagnosis. Even from this one example it is obvious that typical cholelithiasis may cause diagnostic difficulty. The situation becomes more complex when we deal with an atypical form of this polymorphic disease setting in with strange symptoms, which may appear under the mask of a meningitis, an endocarditis, a pulmonary abscess, cancer of the stomach or intestinal obstruction.

The statistics of errors in the diagnosis of this affection disclose so many that it is evident we can only avoid confusion by the most careful investigation of individual symptoms. This necessitates the examination of the patient from head to foot, with the aid of all our modern methods; for there is obvious danger that a symptom which we have carelessly overlooked upon superficial investigation might have been decisive in diagnosis.

The most conspicuous symptom which, from the first moment of a cursory inspection, forces itself upon the attention of the investigator is *jaundice.*

Its diagnostic importance *clears up* the cases which, for a time, have been regarded as ulcer of the stomach, as cardialgia, intestinal colic, or wandering kidney; in these the appearance of jaundice alone reveals the true nature of the malady.

Jaundice in cholelithiasis is either the direct consequence of gall-stones or enlargement of the lymph-glands at the porta hepatis, or it arises independently of these by an accompanying substantive catarrh of the mucous membrane, by neoplasms of the stomach or of the duodenum, by tumors of the pancreas, or by other tumors adjacent to the liver, for example, hydronephrosis.

Jaundice is usually and most readily explained by biliary stasis due to obstruction of the choledochus or hepatic duct by stone, therefore, as Riedel calls it, *pure lithogenous icterus.* The intensity of this jaundice depends chiefly upon the size of the stones, partly also upon the degree of distention of the biliary passages from a preceding passage of stone, and partly also to the position of the stone in a possible diverticulum of the canal. If the biliary passages are dilated at the point where the stone is impacted, or if there is a groove in the stone which permits the bile to flow off unhindered, then, even with very large stones in the common bile duct, the jaundice may be slight or entirely absent; cases such as this were familiar to Morgagni.

With an obstruction by stone in the common duct, icterus may also be absent if an accessory ductus choledochus exists by which the bile may flow into the intestine, a structural anomaly which was observed by Fallopius and Vater. Frank describes a case in which the common duct bifurcated into two branches, one of which emptied into the jejunum and the other into the colon.

Furthermore, jaundice may be absent in spite of occlusion of the common duct by stone, if a simultaneously present gall-bladder-colon fistula permits the bile to flow freely into the intestine; in such cases bile is never found in the vomitus, while the feces, particularly if peristalsis is rapid, show greenish, undecomposed bile which contains bilirubin or biliverdin.

On the other hand, intense jaundice may exist, even with small impacted stones in the common duct, provided secondary catarrh of the biliary passages develops.

Obstructive jaundice and cholelithiasis may also occur from compression of the common duct; either by the gall-bladder filled with stones, or by a dropsical gall-bladder, or by a volvulus and displacement of the same with adhesions to neighboring organs, or by abscesses which have arisen spontaneously in the region of the gall-bladder in consequence of pericholecystitis or after operative procedure.

Diverticula of the gall-bladder and cystic duct containing stone may lead to compression of the common duct, and cause either a permanently deep jaundice or one varying in its intensity.

Besides these forms of lithogenous occlusion, or compression of the common duct causing jaundice, Riedel has familiarized us with another form of icterus, which, in contrast to the purely mechanical form, he has designated *inflammatory jaundice*. This arises by propagation from the inflammatory mucous membrane swelling of the gall-bladder, extending through the cystic duct to the biliary passages. It is at the same time a catarrhal jaundice, but with the difference that in ordinary catarrhal jaundice the process proceeds from the intestine to the common duct, while here the starting point of the inflammatory swelling is the gall-bladder. Riedel bases his justification for the assumption of such a jaundice on the findings at the operation in those cases of irregular and "non-successful" cholelithiasis which have been preceded by typical attacks of gall-stone colic, in which the operation showed only large, round, non-faceted stones in the gall-bladder that could not possibly have passed through the common duct; while, on the other hand, the examination of the feces, which was repeatedly undertaken during the attacks, never revealed the passage of stones.

Unquestionably, provided no stones are found in the common duct at the operation, and compression of the choledochus by stone-containing diverticuli of the cystic duct may be excluded, to this form of inflammatory jaundice belongs the *transitory jaundice* following operations upon the gall-bladder with complete discharge of the stones, which probably originates in the surgical manipulation of the gall-bladder and the cystic duct, apparently by a propagation of the inflammatory swelling of the mucous membrane of the cystic duct to the common duct. Hemorrhage from the damaged wall of the gall-bladder into the biliary passages,

and coagulation of blood in the same, may also cause obstructive jaundice. It must, however, be remembered that the inflammatory changes in the wall of the gall-bladder lead to secondary infection and intoxication by way of the blood current, and these processes may give rise to jaundice which, in patients with gall-stones, may also be looked upon as toxic, and as the origin of the infection in some other point. This is also true of post-operative chloroform jaundice, which differs from obstructive jaundice just as does the previously mentioned septico-toxic jaundice by insufficient intestinal acholia.

Jaundice may vary from the slightest discoloration of the sclera to the most intense icterus. Intense jaundice is always the sign of lithogenous closure of the hepatic or common duct, a cicatricial stricture in consequence of ulceration at the point at which the stone has been arrested. Jaundice in gall-stone colic may develop even in 6 to 12 hours after the attack. This *rapid appearance of jaundice after the attack of pain* differentiates gall-stone colic from those cardialgias which accompany indigestion, and sometimes precede by *several days* the appearance of catarrhal jaundice.

But in cholelithiasis the development of jaundice may exceptionally be also slow and progressive, giving rise to new diagnostic errors by confounding this with neoplasms of the biliary passages which compress the duct slowly and progressively. A chronic, long-existing jaundice, such as is observed after the passage of stone by perforated openings into the intestine and a cessation of the biliary stasis, may clear up quickly.

The rapid disappearance of jaundice, however, does not always indicate a perforation. It may be due to the fact that the impacted stone has become loosened and moves retrogressively in the dilated common duct.

After the passage of the stone into and through the duodenum, a permanent constriction or closure of the ductus choledochus may be produced by cicatricial retraction at the point of perforation, and in this manner jaundice which had cleared up may reappear. In the differential diagnosis between chronic obstructive jaundice due to stones arrested in the common duct and compression of the common duct by stones in the cystic duct or in a diverticulum, the circumstance may be pointed out that, in lithogenous closure of the choledochus, all factors which stimulate the secretion of bile also increase the jaundice, while the same factors, as, for example, when compression of the choledochus is due to a diverticulum, the plentiful ingestion of fluid by surmounting the obstruction, renders the flow of bile into the intestine possible, and, by relieving the biliary passages from the accumulated bile, may cause a decrease in jaundice.

As a well marked example I should like to report a case which I showed in the Clinic upon February 13, 1900, that of a railroad conductor, aged 32, who for four years suffered from gall-stone colic with the characteristic

radiation of pains to the shoulder and chills. In the last attack the patient was permanently jaundiced.

After the appearance of the jaundice the colic did not recur. The icterus, however, remained, and showed the peculiarity that in the winter, especially when the weather was very cold, it was particularly intense, while in the summer it was less noticeable. Appetite and regularity of the bowels were undisturbed. The patient had no symptoms except continuous lassitude. An operation was performed in Albert's Clinic, and showed a stone situated in the diverticulum of the cystic duct compressing the common duct. As with chronic although varying jaundice we usually diagnosticate a lithogenous obstruction of the common duct, the history in this case is notable, particularly the increase of the jaundice in winter and its decrease in summer. This can only be explained by the hypothesis that the patient took more fluid in summer, and, by increasing the biliary secretion, the immobility of the choledochus was overcome. This observation is all the more interesting since, according to Hermann, jaundice due to occlusion of the ductus choledochus increases during a mineral spring treatment at Carlsbad.

In cholelithiasis intrahepatica the jaundice may also depend upon the concentration of bile. It may be entirely absent if the bile is a thin fluid, and, on the other hand, may be intense with an inspissated, thick, sandy, pappy state of the bile (Leichtenstern).

In regard to the development of jaundice in cholelithiasis and, in fact, jaundice in general, those cases are of special significance in which the flow of bile is from the biliary fistula which has been produced by operation, and, in spite of which, jaundice continues when no mechanical obstruction can be demonstrated in the biliary passages.

Inflammatory swelling of the cystic duct was therefore not present, consequently there was no inflammatory jaundice. It can hardly be explained in any other manner than by the view of a *direct lesion of the liver cells*; whether, in Liebermeister's sense, as a diffusive jaundice, or, as Minkowski and E. Pick believe, due to a perverted secretion of the liver cells, cannot be decided at present.

These attempts at explanation coincide with the results of the investigations of Browicz, who refers all forms of jaundice to an increased function of the liver cells, which, stimulated by various influences, take up excessive quantities of nutritive and functionating material, even hemoglobin, and transform them into bile.

The circumstance that jaundice sometimes develops to its fullest intensity even a few hours after the onset of the gall-stone colic, while experimental jaundice after tying the common duct requires at least 24 hours, and usually 2 to 3 days or even longer, led Pick, in the year 1895, contrary to current opinion, to declare that jaundice in gall-stone colic was not obstructive jaundice, but was reflex, due to the nerves of secretion

of the liver (*nervous paracholia*), a reflex conveyed from the sensory nerves of the gall-bladder to the nerves of secretion.

The rapidity with which jaundice develops after the beginning of the attack may also be explained by a mechanical theory, as, in fact, is done by Gerhardt in his lecture upon icterus duodenalis in which he says: "In gall-stone colic the bile in the biliary passages is not only under the pressure of secretion, but under that of the pathologically contracted gall-bladder; therefore, it passes very rapidly into the hepatic veins; 6 hours after the beginning of the attack, traces may be found, and, after 12 hours, there is a well-developed form of jaundice." These contractions of the gall-bladder, however, can only be decisive for the development of jaundice when the cystic duct is permeable, therefore in ordinary cholelithiasis with the passage of the stone through the cystic duct; while in the irregular forms of cholelithiasis with occlusion of the cystic duct, jaundice cannot be thus explained unless we attribute to the substantive reflex contractions of the common bile duct and especially of the circular terminal muscle (*sphincter choledochi*) at the duodenal end the same rôle which we ascribe to the gall-bladder itself. Thereby the pressure of the biliary system exerts a degree of contraction sufficient for the propulsion of the bile into the circulation.

We see, therefore, that a satisfactory explanation of jaundice in all cases of gall-stone colic has not yet been given, and that in its production several factors are probably coöperative.

A symptom which is intimately connected with jaundice is *pruritus cutaneus*, which sometimes precedes the attack or follows it. With an existing jaundice, an increase of pruritus may announce an approaching attack. This symptom, markedly disagreeable, by causing scratching of the skin, may be the immediate forerunner of serious complications, such as furunculosis and secondary staphylococcus infection of the biliary passages, a condition which I saw in a patient from Salonica.

Local cutaneous jaundice in the form of a *greenish-black discoloration of the skin* in the right hypochondrium was observed some time prior to rupture of an abscess which had been formed by a perforation of a gall-bladder containing stones.

Xanthoma are not rare in jaundice; nevertheless I have frequently seen xanthoma in the *canthus of the eye or the lids* of gall-stone patients who had no jaundice, a finding to which I desire to attach a certain importance, particularly in the diagnosis of irregular cholelithiasis.

Symptoms on the part of the nervous system play an important rôle in cholelithiasis, and may appear during the paroxysm as well as in the separate phases of the disease, and may become prominent. It is self-evident that the nervous symptoms may vary according to whether we are dealing with a neurasthenic or hysterical individual, with a child or with an aged person.

Marked psychical disturbances, attacks of mania, general convulsions, confusions and hallucinations, clonic spasms which begin in the muscles of the right side of the abdomen and extend to those of the extremity or affect the muscles at the back of the neck, and symptoms of hemiplegia may occur during the attack and so completely dominate the picture that, in exceptional cases, they may be the cause of death.

The *position of the patient* during the paroxysm may vary; some lie upon the back, others upon the side or the abdomen, others are bent over or kneel, propping the head upon the floor, and others again resort to a cradle motion resembling a mother nursing her child.

The patient that I saw showed during the attack the characteristic "arc de cercle" with hyperesthesia of the skin in the hepatic region and hysterical contractions.

Reflex paralyses, such as occur after affections of the bladder and uterus, were noted by Trousseau as sequels of gall-stones.

Headache is frequently mentioned in the literature of gall-stone disease. Frerichs observed cases in which marked headache, cardiac palpitation, active pulsation of the abdominal aorta, and epistaxis became prominent accompaniments of the attacks. Kehr saw cases in which migraine disappeared after the removal of gall-stones. Kraus found migraine a feature in the history of many gall-stone patients, and remarks that in some of these, on the first appearance of gall-stone colic the attacks of migraine became rarer or ceased entirely. I, too, have seen cases of the latter kind, but I believe that in most of them the finding of the two frequent pathologic conditions is a mere coincidence.

Headache in chronic jaundice is sometimes a phenomenon of cholemia; in other cases it is due to anatomical disease of the meninges, as, for example, pachymeningitis hemorrhagica or meningitis, which latter affection is not infrequently combined with infectious cholangitis, and forms the terminal phase of gall-stone disease. Therefore, in the course of a cholelithiasis, we may meet with severe cerebral symptoms, with delirium, with sopor and stupor. On this account, cholelithiasis is intimately related in a differentio-diagnostic respect to acute atrophy of the liver, in which the early appearance of psychical disturbances out of proportion to the other symptoms forms a striking diagnostic factor. Years ago, when an assistant in Bamberger's Clinic, I was called to Braun's Obstetrical Clinic to see a pregnant woman who was suddenly attacked by severe pain in the hepatic region accompanied by jaundice, so that at first she was thought to be suffering from gall-stones. Soon afterward delirium set in and the liver dulness progressively decreased. The autopsy showed acute yellow atrophy of the liver. Decrease of liver dulness in pregnancy is by no means easy to determine. Delirium and psychical disturbance may be observed in both diseases, and, besides, a chemical analysis of the urine for leucin and tyrosin cannot be immediately made at the bedside, nor is

such test absolutely decisive, for cases of cholelithiasis have been observed in which leucin and tyrosin were present in the urine and were apparently due to focal necrosis of the liver cells.

Among the nervous symptoms, the *pains* which accompany the paroxysm of gall-stone colic form the most prominent symptom in the clinical picture. They begin usually 3 to 4 hours after a meal, therefore when the chief bulk of the acid food mass is passing into the duodenum, in contrast to the cardialgias of gastric ulcer and acute catarrh which appear soon after the intake of food. Owing to the active peristalsis of the biliary passages, the stone is caught in the neck of the gall-bladder, and is thus forced further into the cystic duct. When the spasm of the musculature of the gall-bladder ceases, the stone may regurgitate into the bladder, but, as a rule, it is carried onward until it finally reaches the duodenal mouth of the choledochus, and, eventually, the intestine. This transit of the stone from the gall-bladder to the duodenum in typical cholelithiasis usually runs its course under the well-known picture of gall-stone colic. The pains are here due to an acute dilatation of the biliary passages and irritation of their sensory nerve fibers, either by the stone or by the inspissation of bile caused by the stone, or also to an erosion of the mucous membrane by a stone with sharp edges.

Exacerbations of pain in an acute irritation of the biliary passages are caused by contractions of the musculature of the gall-bladder and of the biliary passages, analogous to uterine contractions in the period of expulsion. The greatest obstruction to the passage of stones is formed by the narrow cystic duct and the portio intestinalis of the choledochus.

The *two phases of pain* which theoretically are said to occur in the passage of the cystic duct and of the duodenal papilla in typical cholelithiasis, and which are not to be undervalued as diagnostic factors as two entirely distinct exacerbations of pain in the clinical picture of cholelithiasis as it has been described by some authors, may occur, but I have not as yet observed them in my cases. The cessation and the exacerbations of pain are due to contractions, and I believe that, analogous to rapidly succeeding uterine contractions, it is subsequently impossible to determine whether the first, the middle, or the last of the paroxysm was the most painful. I also believe that, in cholelithiasis, the line of demarcation between these two periods of pain, which is said to mark the passage of the stone from a narrow into a wider canal, is more theoretic than real.

According to Riedel, the paroxysms of pain in cholelithiasis, also the less marked pain at the onset of the attack, are not to be attributed to the impaction of stone in the cystic duct but to *cholecystitis*. The *vis a tergo*, which forces the stone into the cystic duct, is the pressure of the rapidly oncoming inflammatory exudate in the gall-bladder (*inflammatory dropsy of the gall-bladder*).

Riedel even attributes the colics which recur after the impaction of the stone to renewed inflammation of the gall-bladder.

The attacks of colic which accompany lithogenous closure of the cystic duct are designated by Riedel as *ineffective* if the stone does not pass the cystic duct, and are in contrast to *effectual* colic in which the stone, after having passed the cystic duct, enters the common duct, and finally reaches the duodenum.

Similarly, Riedel considers the attacks of colic including the jaundice which appears after the stone has reached the supraduodenal portions of the choledochus without occluding them and without producing stasis of bile, to be caused by a sudden inflammatory reaction of the mucous membrane to the foreign body.

We see, therefore, that even the mechanism of gall-stone colic is susceptible of different explanations.

In cases of cholelithiasis in which the colic manifests itself only by gastric spasm, vomiting and sensitiveness to pressure in the region of the gall-bladder, the exacerbations of pain upon movement in opposition to pain of other origin may have diagnostic importance.

A patient in the Clinic with cholelithiasis complained of exacerbations of pain when riding in poorly paved streets, and when jolting during the drive to the hospital, this being analogous to the onset of the pain in patients with stone in the bladder; another patient, who besides sensitiveness of the gall-bladder upon pressure also had friction sounds in the same region, remarked that upon using the right foot in jumping the pain in the gall-bladder region and above the right Poupart's ligament was particularly severe. Upon jumping on the left foot painful sensations were felt only in the gall-bladder region, and this was the case in rapid running or when lying upon the right side. In ascending a staircase the pains were more severe than in descending, but they were especially severe in jumping upon the right foot. The vertebral column, from the second to the sixth thoracic vertebra inclusive, was painful upon pressure, and within this zone of pain cutaneous hyperesthesia was present and more marked upon the right than upon the left side.

The *radiation of the pain* in cholelithiasis demands special notice. In classical cases, the pains begin in the region of the gall-bladder and radiate to the right shoulder. At the onset of the attack, some patients experience a peculiar chilliness and a drawing sensation along the back. Combined with this the region of the gall-bladder is sensitive to pressure. In contrast, there is quite an array of cases in which the localization of pain is an irregular one, and the pain radiates toward various regions of the body. The pains may be localized immediately below the xiphoid process without radiating toward the hypochondriac region, and without increasing upon pressure. Confusion with pretubercular gastralgias, dyspepsia, ulcer and cancer of the stomach, and gastric crises is therefore possible.

Sometimes the pains are localized in the breast, in the thorax, in the œsophagus, sometimes in the region of the mamma, so that the patients believe they are suffering from cancer of the breast. When the pain radiates to the abdomen, particularly around the navel, intestinal colic and enteralgia are simulated. The diagnosis between saturnine colic and gall-stone colic may, under some circumstances, cause difficulty, as in a case which came under my notice, occurring in a worker in artificial flowers; the patient simultaneously showed a lead line and mild jaundice. In women, affections of the genitalia, metritis, parametritis and the like are suggested to us, all the more so as gall-stone colic sometimes appears during menstruation. Frequently appendicitis comes into consideration, particularly if the vermiform process is bent upward or is even adherent to the gall-bladder.

The pains are stabbing or tearing; sometimes there is a girdle sensation. They may also wander with a change of position. The colicky pains in the right hypochondrium may be quite transitory or may last for several days. Radiation to the back or to the lumbar region, as occurs in carcinomata of the pancreas, in ulcer of the posterior wall of the stomach, in carcinoma of the stomach with metastases of the retroperitoneal glands, and in ulcer and carcinoma of the duodenum, is also observed in rare cases of cholelithiasis. In this affection the sensitiveness of the lumbar and dorsal vertebræ to pressure and to percussion is similar to that observed in ulcer of the stomach. But pain upon tapping the vertebra with the fingers appears to be more readily induced in gall-stone disease. In a patient, aged 68, added to the typical hepatic colic were burning pains radiating into the lumbar region and the region of the urinary bladder, with strangury and the discharge of dark urine depositing a profuse, red sediment. The patient suffered formerly from rheumatism of the joints of the hand and of the ankle-joints.

[Tenderness on palpation in the region of the gall-bladder, due to cholelithiasis or to cholecystitis, is characterized by a radiation of pain to the epigastrium. This fact I have observed in my own case and in many patients. Tenderness upon palpation in the right hypochondrium due to other causes may radiate or may not, but is not felt in the epigastrium.—Ed.]

In such cases, particularly if jaundice is absent, gall-stone colic is apt to be confounded with renal colic.

Irradiation of pain to the breast may be confounded with pleurodynia, rheumatism and intercostal neuralgia.

In the differential diagnosis of gall-stone colic and *nervous hepatic colic*, Th. Fuchs remarks tersely that nervous hepatic colic is invariably aggravated by a Carlsbad treatment. I have not as yet seen such an instance. The majority of cases which, upon superficial investigation, were supposed to be hepatic colic, proved later to be intercostal neuralgia. Some

cases, too, of hysterical pseudo-appendicitis were found upon minute investigation to be nothing more than neuralgia of the 12th intercostal nerve and its ramus perforans anterior. In such cases the painfulness of the vertebral and lateral pressure points, as well as the presence of other nervous symptoms, and the hysterical stigmata are decisive.

In those cases in which the pain radiates toward the cardiac region and toward the left arm, the diagnosis may become very perplexing and even impossible. I observed a case of this radiation of the pain in a patient about 50 years of age, who, for some years, suffered from slight paroxysmal glycosuria, uraturia and oxaluria. Suddenly the pain appeared in the gall-bladder and thence radiated, the amount of sugar in the urine was increased to 4.5 per cent., the pulse rate declined to 45. Cardiac examination was negative except for a moderate accentuation of the second aortic sound. The diagnosis of competent physicians varied between angina pectoris, as a consequence of sclerosis of the coronary arteries, and cholelithiasis. The patient consulted me several months after the attack, and I made a diagnosis of gall-stones, chiefly for the reason that the gall-bladder was sensitive upon pressure, that the patient showed xanthomata bilaterally upon the eyelids, and because the glycosuria which had been observed after the attack but had now improved so that only traces of sugar were present could be best reconciled with the diagnosis of cholelithiasis. A marked increase in the excretion of sugar in angina pectoris of the diabetic is unknown to me.

In contrast to this I remember a case in Bamberger's Clinic, occurring in a woman, aged 35, who suffered from aortic insufficiency with coronary angina (angina pectoris). After such an attack, which ran its course with vomiting and slight jaundice, sensitiveness upon pressure over the liver appeared. The diagnosis was insufficiency of the aortic valve, sclerosis of the coronary arteries, cholelithiasis. The patient died soon afterward during an attack; the autopsy showed insufficiency of the aorta, narrowing of the coronary arteries, but *no* gall-stones.

These two cases prove that we may meet with the serious dilemma of being doubtful *whether angina pectoris or cholelithiasis* is present, and in this case every objective sign which may be of importance in the differential diagnosis between these affections must be minutely investigated.

Colic-like intestinal pains in the hypochondrium and right hypogastrium, with meteorism and constipation, may occur in cholelithiasis and be due to adhesion of the gall-bladder to the transverse and ascending colon. The development of adhesions of this kind is especially favored by a pendulous belly and hepatoptosis. Tearing and drawing pains in the hepatic region before each movement of the bowel, and pain radiating to the right shoulder, were observed by Kelling in a case of cholelithiasis in which the transverse colon formed adhesions with the border of the liver. After breaking up the adhesions, the pains disappeared.

Typical attacks of colic with chronic jaundice may be due to *simple adhesions without gall-stones*. In such a case, reported by Leichtenstern, the entire gall-bladder was atrophic, and firmly adherent to the surrounding tissues; no stones were present, neither in the cystic nor in the common duct. The adhesions were broken up, the jaundice disappeared soon after the operation, and the patient was completely cured.

Unlike many forms of pain arising in saturnine colic, the pain is usually unrelieved by pressure. Sometimes there is amelioration after the eructation of gas or after the passage of flatus and feces. Such cases may be confounded with hepatic colic and flatulent colic.

In the majority of cases the ingestion of food and fluid does not specially influence the pain. Though it sometimes happens in gall-stones that the attack of pain develops immediately after the intake of food, and erroneously leads to the diagnosis of ulcer of the stomach, and although, on the other hand, in the case of an ulcer with decided secretion of gastric juice and hyperchloria, not rarely the pain ceases after the ingestion of food, whereby the HCl is combined, yet the influence of the character of the food and the fluid ingested must not be undervalued. Patients who formerly experienced no difficulty in taking food which was highly spiced and seasoned now find themselves unable to partake of this without severe gastric pain; this circumstance, in a given case in which the diagnosis hinges between ulcer and cholelithiasis, decides us in favor of ulcer, as in a case I observed, which, by the later occurrence of hematemesis, proved to be ulcer. The pain is sometimes augmented after drinking sour wine, and this may, under some circumstances, favor ulcer, while a patient with gall-stone, who was known to me, after taking quite a quantity of wine, noted amelioration of her pains.

Gall-stone patients can sometimes digest heavy and irritating foods very well, but there may be idiosyncrasies for certain foods. A patient whom I knew, according to his account, invariably had an attack of gall-stone colic after eating fish with mayonnaise; some drugs, pepsin and hydrochloric acid, administered for gastric difficulty of doubtful origin, may be a guide in the diagnosis of these cases which are so frequent in practice. While I have seen no noteworthy influence upon the symptoms of cholelithiasis after the administration of pepsin and hydrochloric acid, I saw a case in which this remedy was given for cardialgia; the pains increased and a few days after the use of the remedy hematemesis occurred. This observation led us to be more cautious in the use of an active pepsin in doubtful cases; and it may be noted that even such a therapeutic experiment, which was here accidental, may be of value in those which are obscure.

The nocturnal exacerbations of pain which occur in a great number of cases of cholelithiasis are explained by Kehr as being due to the accumulation of bile in the gall-bladder. In biliary fistula we observe that

the excretion of bile during the day is very slight, but it is profuse during the night. "During the night when a person sleeps and no food is taken, bile collects in the gall-bladder." Although this remark and explanation of Kehr are quite correct, and may be utilized in the differential diagnosis between gastric ulcer and cholelithiasis, inasmuch as pain from ulcer is rarely present in an empty stomach or at night, while gall-stone colic frequently occurs at night with an empty stomach, no absolute proof can be attached to this symptom, the less so as pains, particularly at night (Hesse) have been observed in ulcer, as well as amelioration of the pain on the ingestion of food, especially when there is simultaneously existing hypersecretion. The nocturnal appearance of pain is emphasized in the rare syphilitic form of gastric ulcer.

I have not tried the effect of a *hunger cure* carried out systematically for several days in suitable cases for diagnostic purposes, although it is reasonable to assume that such an attempt is justified in complicated cases, especially when the question of operation is under consideration. *A priori*, it must be taken for granted that, for example, the gastric crises of the tabetic are uninfluenced by such a hunger cure; the same is very likely true to some extent of pains due to stone in the biliary passages, while in ulcer, because of insufficient gastric secretion in the overwhelming majority of cases, the pain either ameliorates considerably or ceases entirely.

The variable *intensity of the pains* in cholelithiasis is not always in proportion to the other reflex symptoms on the part of the stomach, for example, vomiting. In a patient observed in the Clinic, a day laborer, John Seh., aged 57, the paroxysms of pain in the right, upper abdominal region, running their course with jaundice, were of such extraordinary severity that the patient writhed in bed, moaning. In this severe paroxysm of pain which, according to the accounts of the patient, was much more severe than the earlier ones in which vomiting occurred, symptoms on the part of the stomach were entirely absent.

The pains which are due to pericholecystitis, perihepatitis, circumscribed abscesses or rupture into the peritoneum, belong to the realm of *peritonitis*.

It must not be forgotten that abdominal pain and pain in the right hypochondrium during menstruation may also be due to gall-stone colic. The same is also true in pregnancy.

Cases have been reported in which, at the normal termination of pregnancy, gall-stone or renal colics have been mistaken for labor pains; also in childbed, when pains arise in the abdomen with vomiting, fever, and chills, cholelithiasis may be simulated. Here only an exact history, the appearance of pain a few hours after meals, the pulse, and a most thorough examination of the gall-bladder region and the right hypochondrium will lead to the diagnosis of a complicating cholelithiasis.

The *differential diagnosis between gall-stones and pancreatic stones,* especially as jaundice may occur also in the latter affection, causes great perplexity.

Simultaneous diabetes, or that occurring in combination with colic, fatty stools, azoturia, and, finally, the passage of stones of calcium carbonate and phosphate, are important diagnostic points of support.

When *tabetic gastric crises and jaundice occur simultaneously,* the diagnosis is sometimes only possible by the careful discrimination of the individual symptoms.

I saw a patient who for four years suffered from attacks of pain which occurred irregularly at intervals of from three weeks to three months; the patella tendon reflex was absent; there was immobility of the pupil. During an attack a year ago jaundice lasted for two weeks. A person suffering from tabes may, therefore, simultaneously have cholelithiasis, or, by a temporary indigestion, catarrhal jaundice may be superadded.

Prior to this we have considered two of the most frequent and marked symptoms of cholelithiasis, *jaundice and pain.* Just as there are cases of cholelithiasis without jaundice, there are others, with or without jaundice, which run their course with freedom from pain. Cholelithiasis without pain is certainly a very rare form. Complete but painless occlusion of the choledochus by stone may appear under the guise of catarrhal jaundice or of a neoplasm at the porta hepatis. In chronic empyema of the gall-bladder, local pain may be very slight or entirely absent.

In these *indolent* forms of cholelithiasis, another element becomes prominent and almost substitutes for the attacks of colic, namely, *fever,* which, therefore, may substantively be of great diagnostic significance.

In the *aged,* in whom attacks of gall-stone colic need not necessarily cause serious disturbance, a mere chill may be the only manifestation of the disease.

In other cases, chills may alternate with pain in the right hypochondrium. The chill may run its course with collapse, cyanosis of the lips and cold extremities, followed by fever with or without sweating. The paroxysms of fever in cholelithiasis begin, as a rule, a few hours after eating, therefore in the evening or during the night, in contrast with those of malarial attacks.

Even when the stones are large and completely obstruct the common duct, this intermittent fever may dominate the clinical picture. As an assistant in Bamberger's Clinic, I once saw a patient with obstructive jaundice and intermittent fever, who, according to her report, had *never* suffered from colic, and who, upon *objective* examination, showed no local sensitiveness in the hepatic region; at the autopsy, a stone the size of a walnut obstructing the common duct was found, while no signs of a preceding lithiasis were present in the gall-bladder. Bamberger regarded this case as a secondary gall-stone formation in the common duct, developed

by the apposition of the cholesterin to a stone which had wandered into the common duct without giving rise to symptoms, and the painlessness of its course as due to the mucous membrane becoming accustomed to the foreign body slowly growing *in situ*.

The *onset of the pain with chills* is important in the diagnosis between gall-stone colic and cardialgia in ulcer of the stomach. Rises in temperature in ulcer of the stomach may be caused by circumscribed peritonitis. The preceding gastric disturbances, the dependence of pain upon a co-existing peritonitis, the absence of chills will also aid in the diagnosis of such complicated cases.

Doubt may arise in the differentiation of *duodenal ulcers* which develop upon the basis of a *paroxysmal hemoglobinuria*. An interesting case of this kind was admitted to the Clinic in May, 1895, with a diagnosis of ulcer of the stomach. The patient was always well until July, 1892, when he was taken ill after having been for two days previously exceedingly pale, and, according to the statement of his wife, the sclera having become yellow. During a journey he was attacked by nausea, severe gastric pains, vomiting of greenish masses, and collapse without loss of consciousness. The vomiting was repeated once, but did not recur. On the other hand there was gastric spasm, loss of appetite, acid eructations and a feeling of heat; the bowels were constipated. Melena and hematemesis never occurred; the urine was blood red in color. A few days after the patient had left his bed, he completely recovered.

Up to January, 1895, the patient had three such attacks. In the last, which caused him to come to the Hospital, there was pallor and loss of appetite, the gastric pains recurred and radiated toward the lumbar region to the right of the navel. He vomited greenish masses of an acid odor; there was pyrosis and eructation. Acid food increased the gastric distress, while the pains were lessened by sodium bicarbonate. The objective examination showed high-grade anemia and a moderate degree of gastric dilatation. The majority of the symptoms, therefore, indicated ulcer of the stomach, and two only were calculated to obscure the diagnosis: On the one hand, the chill which actually introduced the first attack, and, on the other hand, his wife's report of the yellow discoloration of the sclera in the first attack. These two symptoms favored cholelithiasis. Nevertheless, our tentative diagnosis was: Ulcer of the stomach.

The patient left the Hospital. Four months later he returned to the Clinic with the same symptoms, and, in addition, a nephritis and hemoglobinuria. The attack was introduced by a chill. Several months later the patient died. The autopsy showed *ulcer of the duodenum*. We see, therefore, that with the symptom-complex obtained by the history—cardialgia, chills and jaundice—instead of the looked-for gall-stones, an unexpected combination of two rare affections prevented a correct diagnosis. In the majority of cases, however, the occurrence of rare cases of this

kind is not calculated to lessen the clinical value of the previously mentioned triad of symptoms in the diagnosis of cholelithiasis.

The chills which accompany fever and gall-stone colic were formerly believed to be reflex and due to the influence of the heat-regulating center. On this conception of fever as a pain reflex, Bamberger chiefly based his opinion of the favorable action of morphin, which, in some cases, by relieving the pain, simultaneously had an antipyretic effect. In many other cases, however, we are compelled to assume a pre-existing, or subsequent, infection of the biliary passages. This is true, in particular, of those cases in which the febrile paroxysm is not combined with an attack of pain, and even more so in those instances in which the febrile attack is the only manifestation of cholelithiasis. Infection of the biliary passages may occur in various ways, one of which is that the bacteria find a passage directly from the intestine into the common duct. If, by disease of the biliary passages, a *locus minoris resistentiæ* is formed for the entrance of bacteria, the latter, on the other hand, is favored by dilatation of the biliary passages and the opening of the duodenal mouth, and thus infection with intestinal bacteria takes place. There is also the possibility of infection with virulent bacteria from the oral and pharyngeal cavities, for example, from tonsillar abscesses the discharges of which are swallowed with the saliva. Infection of the biliary passages may also occur via the circulation from distant foci in the organism, as from otitis media and angina tonsillaris, etc., as is also assumed to be the case in appendicitis. These manifold sources of infection also explain the bacterial polymorphie nature of infectious cholecystitis and cholangitis (bacterium coli, diplococci, streptococci, staphylococci and typhoid bacilli).

The attacks of fever which accompany paroxysms of pain in cholelithiasis are, at present, almost universally referred to infection of the biliary passages, and are not considered as reflex fever, as was formerly the case. Although we must admit the possibility that a typical colic fever in which chills, fever and paroxysms of pain go hand in hand may be a nervous reflex fever, analogous to the febrile attacks in paroxysmal hemoglobinuria, or a purely toxic fever due to pyrogenous derivatives, perhaps of hemoglobin, nevertheless, it cannot be denied that, in the overwhelming majority of cases of *intermittent hepatic fever* infectious processes in the biliary passages and in the liver are the causative factors. This is particularly true of the cases in which fever appears in a substantive form and independent of the colicky pain, and further, of those cases in which there are objective signs of a pericholecystitis or perihepatitis, and, finally, when we have, as accompanying symptoms of the fever, signs of a local inflammation or infection in other organs removed from the liver, or in the blood, as, for example, enlargement of the spleen, nephritis, endocarditis, pericarditis, bacteriemia and septic leukocytosis.

There are many transitional stages between the periodic intermittent

hepatic fever and the severest intrahepatic infectious processes, hence it is clear that the differential diagnosis, whether intermittent hepatic fever, or pyemic fever in consequence of an ulceration, or cholangitis which has caused abscess, or pylephlebitis, can only be arrived at, in the given case, by the most minute investigation of the patient's entire condition. Intermittent fever in consequence of cholangitis is not rare in obstructive jaundice. It is true it is more frequent in occlusion of the common duct due to stone than, for example, in carcinoma at the porta hepatis. It may occur in cholelithiasis without jaundice by the propagation of the infection from the gall-bladder to the biliary passages, or, with an existing gall-stone formation, in the branches of the hepatic duct. Intermittent fever, with or without jaundice (the latter of which so frequently occurs in cholelithiasis intrahepatica), may occasionally cause great diagnostic perplexity if, after the gall-bladder has been extirpated, we must decide the important question of the treatment which is to follow (whether a cure at Carlsbad or another operation), whether the stone has formed in the operated stump of the cystic duct or in the branches of the hepatic duct. This secondary cholelithiasis intrahepatica may, therefore, make the result of a successful extirpation of the gall-bladder illusionary, may prove that the intermittent fever existing before the operation was the initial sign of an intrahepatic gall-stone formation, and will necessitate caution on the part of the surgeon in the choice of his operation (cholecystostomy, drainage of the hepatic duct). In contrast to diffuse cholangitis, which sometimes runs its course with high temperature but without profound changes in the walls of the biliary passages, a localized choledochitis from impaction of stone with ulceration of the duodenal portion of the choledochus may also be the cause of the fever, in that, from the point of the stone up to the diverticulum of Vater, a portion remains like a blind sac communicating with the duodenum which is not touched by bile. *In this dead angle, similarly as in the appendix,* bacteria may collect and produce ulceration with general sepsis. By a propagation of the ulcerative process to the portal vein, pylephlebitis and pylethrombosis may develop. The diagnosis of purulent cholangitis or suppurative pylephlebitis with abscesses of the liver may be extremely puzzling. But, in cholangitis, the individual attacks are frequently separated by afebrile intervals, and during these the pulse is more quiet, and there is relative euphoria. In pylephlebitis, on the other hand, chills frequently recur several times a day, the thermometer shows steep curves of abnormally high rise and subnormal fall. High pulse rate and marked collapse point to pyemia.

Furthermore, in pylephlebitis diarrhea becomes prominent, there is marked enlargement of the spleen, metastases form in the lungs and other portions of the body, and, finally, there is, besides, occlusion of the portal vein.

Abscesses of the liver may also be produced by *actinomycosis.* We

observed in our Clinic a classic example of such actinomycotic abscess of the liver following appendicitis actinomycotica and metastatic miliary actinomycosis of the lungs; there was sensitiveness of the liver upon pressure, chills and pyemic fever.

Intermittent fever is not always produced by infections or purulent proeesses, especially in the biliary passages, the liver, and the branches of the portal vein. A non-purulent peripylephlebitis intrahcpatica chiefly characterized histologically by wandering cell infiltration around the portal vein branches in the liver may, similarly, run its course with intermittent fever, as was proven by a casc in our Clinic.

The same is trne of *tuberculosis at the porta hepatis,* which may appear under the mask of cholelithiasis. In a servant, aged 43, in whom no hereditary disease was evident, and who was treated for intermittent fever and severe pain in the hcpatic region, sometimcs accompanied by chills and severe vomiting, cholelithiasis was at first diagnosticated; but the later course of the affection, with increasing emaciation, night sweats, signs of infiltration at the apcx of the lung, and tubercle bacilli in the sputum, showed the true nature of the malady. The autopsy revealed tubcrculosis of the glands at the porta hepatis, tuberculosis of the pulmonary apices; no gall-stones.

Intermittent fever in carcinoma of the liver is not rare. In the year 1894, in our Clinic, there was a cachectic patient with typical tertian intermittent fever, in whom, about midday, rises in temperature to 104° and 105.8° F., but without chills and sweating, recurred regularly. An examination for malarial parasites was negative. The liver was enlarged, hard, and showed coarse nodules. A diagnosis of carcinoma of the liver was made, and was confirmed by the subscquent course. In connection with this I should like to mention another case which soon afterward presented itself in the Clinic. This occurred in a patient whose liver was hard and nodular, the spleen cnlarged, and in whom, besides, there was hard, indolent, glandular enlargement of the neck. This patient also had intermittent fever. Our diagnosis at first was carcinoma of the liver with metastases in the glands of the neck. As, however, the affection in its progress showed no conspicuously malignant character, we concluded to administer potassium iodid, and to employ intravenous injections of corrosive sublimate.

During this treatment the enlargement of the liver, especially its isolated nodules, as well as the swelling of the glands of the neck, decreased in size, the fever disappeared, and, in consequence, we changed our diagnosis to syphilis of the liver and the lymphatic glands. This compelled us to bring into relation with each other the intermittent fever which disappeared under antisyphilitic treatment and the underlying process, syphilis.

Moreover, *intermittent fever is also associated with adhesions of the*

gall-bladder, yet no stones are found at the operation. Gersuny performed an operation on the wife of an apothecary who had irregularly recurring colic, chills with fever, enlargement of the liver, pain in the region of the gall-bladder, splenic tumor and gastric dilatation. He found the gall-bladder contracted, no cholangitis, no stone; but the gall-bladder was adherent to the duodenum, and there was gastric dilatation. After breaking up the adhesions the fever disappeared and recovery took place. The process may, perhaps, have been due to retention of bile in the adherent gall-bladder, which formed a favorable culture media for the microbes there present. By the succeeding restoration of free communication between the gall-bladder and the cystic duct, the condition was relieved.

As a cause of the development of intermittent fever, chronic morphinism is yet to be discussed. A case of this kind is known to me in a morphin habitué in whom the most minute investigation of the various organs revealed no cause for the fever. Examination of the urine showed acetone and a conspicuous increase of uric acid excretion to 40 grams per day. In gall-stone patients who, in consequence of preceding colic, have become morphin habitués, this possibility must be borne in mind, and an examination be made of the urine, which, in hepatic fever, shows a decrease in the excretion of nitrogen.

The foregoing remarks prove conclusively the many-sided nature of intermittent fever, and how easily errors in diagnosis may be made in cases of cholelithiasis without pain or jaundice.

All infections of the biliary passages are not accompanied by fever. Even severe cholangitis and empyema of the gall-bladder with threatening perforation may run their course entirely without fever and pain, particularly in marantic individuals and diabetics. Hypothermia is observed particularly in infections by the bacterium coli.

If, after describing the cardinal symptoms of gall-stone disease, either isolated or combined with other affections, we proceed to the *examination of the different organs of the body,* we will in some cases meet with a symptom which is noteworthy in prognosis as well as in regard to the proper therapy to be employed. *Epistaxis* may be the first signal of the *hemorrhagic diathesis* due to the obstruction of bile excretion, but it may also occur in cases in which there is no occlusion of the choledochus with complete stasis of bile and jaundice as a consequence of severe damage to the hepatic cells, and thus complicate the clinical picture. Acute, subacute and chronic infections of the biliary passages and, particularly, secondary cirrhosis, may cause a peculiar predisposition to hemorrhage which influences the clinical picture in a dangerous manner, and may be especially troublesome to the surgeon. Even after jaundice of brief duration hemorrhages appear from the skin, the gums, the nose, the uterus, the urinary passages, the stomach and the intestines, which, inasmuch as the wound

produced by the operation shows no cause for the condition, necessitates the utmost caution on the part of the surgeon.

A dancer, aged 22, was admitted to the Hospital—special stress must be laid upon her occupation, since, in the etiology of cholelithiasis, a sedentary habit is considered to be predisposing—after an undoubted cholelithiasis with alternating jaundice which had existed for seven weeks; there were cutaneous hemorrhages upon the hands and feet, and after scratching off a wart ad nates, a hemorrhage occurred which could scarcely be controlled.

In an examination of the neck, the *jugular glands* must not be overlooked since they are of importance in the differential diagnosis of carcinomata of the biliary passages and of the liver. It is sometimes advantageous to have the patient cough during the examination. Deeply situated, isolated glands behind the clavicle become prominent during coughing, and are thus made susceptible to palpation.

Examination of the lungs is important inasmuch as, according to some authors, tuberculosis predisposes to the formation of gall-stones. In my experience, however, clinically well-developed cholelithiasis, as well as cholangitis, is an extremely rare complication of tuberculosis, and this is all the more remarkable as, in ulcerative phthisis, bacteriemia (streptococcia and staphylococcia), which is so very frequent, furnishes a fertile soil for infection of the biliary passages. I have found, on the contrary, that the tubercular have a certain immunity from gall-stone formation, and patients with cholelithiasis from tuberculosis.

Attacks of cough occurring in paroxysms (the *tussis hepatica* of the older authors) followed by aphonia, hoarseness and severe pains in the back of the neck, have been observed in cholelithiasis and have disappeared after operative removal of the stones.

Dyspnea, sensations of fear, even attacks of suffocation, also occur as symptoms of gall-stone colic, and may be due to reflex spasm of the muscles of respiration.

Immobility of diaphragmatic respiration, therefore, the costal respiration type, in adults, particularly in men, is an important symptom in peritonitis, in contrast to the respiration in gall-stone colic; while small children who, as is well known, normally almost exclusively show the abdominal type of respiration, present the costal type of respiration, and this symptom is an important sign in the differential diagnosis of infantile gall-stone colic, in opposition to intestinal colic of other origin. Sensitiveness to pressure over the gall-bladder, especially when in a warm bath, and muscular rigidity on the right side of the abdomen, combined with the preceding symptoms, form additional proof. Cholelithiasis under twelve years of age, and even under fifteen years, belongs, in fact, among the greatest rarities.

In children, provided jaundice does not point to the possibility of gall-

stones, epigastric pain, vomiting and convulsions are many-sided symptoms, and this doubtless will sufficiently explain the fact that the disease is usually overlooked in early childhood.

In the aged, as the result of gall-stone colic, which, as has been stated, sometimes manifests itself only by chilliness and fever, a *congestion of the lower lobe of the right lung* is observed, which, when combined with cough, muco-viscid expectoration and numerous crepitant and fine moist râles at the base of the right lung, also with fever and pain, may be mistaken for pneumonia. This hyperemia of the lungs, appearing under the guise of pneumonia, may clear up in two or three days after the rudimentary attack of colic. It may, however, also form the starting point of a secondary pneumonia. In cases in which cholelithiasis, by infection of the biliary passages, has led to suppurative cholangitis, biliary abscesses of the liver, or subphrenic suppuration, important diagnostic processes may take place at the base of the right lung: unilateral, high position of the diaphragm with retraction of the lung, pleurisy, symptoms of perforation, fetor of the respired air and, a few days prior to rupture, distressing cough with purulent, bile-stained expectoration and râles at the base of the right lung. Nevertheless, rupture of the diaphragm and pleura, as well as subphrenic abscess, with the formation of a pyopneumothorax, supraphrenicus and subphrenicus, are very rare episodes in the course of infectious cholelithiasis, in contrast to echinococcus of the liver, which, as is well known, frequently forces a passage into the lungs by perforation of the biliary channels and of the diaphragm.

In the course of cholelithiasis *symptoms on the part of the heart* and circulation may dominate the situation to such an extent that, in some cases, a gall-stone patient resembles one affected by cardiac disease. Collapse, cyanosis of the lips, and cold extremities during the paroxysms of pain are observed in nervous, hysterical individuals, particularly in women, and may appear also without chill and fever as a vasomotor reflex, or in old persons as signs of cardiac asthenia. Cardiac arrhythmia, dyspnea, gallop rhythm, with accentuation of the second pulmonary sound, dilatation of the right heart, and displacement of the apex beat, relative tricuspid insufficiency, pulsation of the jugular vein with cyanosis, and a small, easily compressible pulse may occur in consequence of an attack of colic and be a serious symptom-complex in a prognostic sense; with an intact myocardium these are considered as reflex, transmitted by irritation from the sensory nerves of the biliary passages to the pneumogastric nerves and to the sympathetic plexus of the cardiac fibers.

The *pulse* in gall-stone colic is of moderate intensity, either normal or slow. Bamberger found it slow and small in the majority of cases; in others again it is small and frequent, especially when there is a simultaneous inflammation of the gall-bladder and biliary channels. A small, frequent pulse, naturally, may also occur in general peritonitis and in gastric and

intestinal hemorrhages, as well as in septic and pyemic fever in gall-stone patients, and during the apyrexia, especially if an infection from the biliary passages and the liver localizes itself in the heart as a pericarditis, endocarditis or myocarditis.

The tension and configuration of the *abdomen* may furnish objective signs of importance in the diagnosis, particularly as regards the complications of cholelithiasis. *Contraction of the abdominal muscles,* mainly upon the right side, which the French designate as défense musculaire, frequently accompanies the attack of colic; but there are also cases in which the abdomen, in spite of severe pain, is everywhere soft, sometimes even showing meteorism. *Meteorism* which occurs in jaundice in consequence of intestinal acholia, or with a co-existing peritonitis in an obstruction of the intestine by stones, or in intestinal stenosis in consequence of adhesions, is easy to explain. *Ascites* may occur in cholelithiasis by occlusion of the portal vein. The irritation of the incarcerated stone in the common duct produces consecutive induration in the hepato-duodenal ligament, and, as a further result, thrombosis of the portal vein and an ulcerative perforation of the walls by which stones enter its lumen.

In this group belongs the case that is everywhere quoted of Ignatius de Loyola, in whose portal vein Realdus Columbus found three gall-stones. In general, however, ascites in cholelithiasis is rare in comparison with carcinoma at the porta hepatis, which, by frequent compression of the portal vein, as well as by secondary carcinosis of the peritoneum, leads to ascites. This is particularly true of the cases running their course with obstructive jaundice, just as an ascites developing simultaneously with jaundice almost always indicates an abdominal neoplasm.

In gall-stone patients the *panniculus adiposis* (subcutaneous fat) is often markedly developed, particularly in the abdomen, which naturally renders the physical examination difficult.

Peter observed, during the attacks of colic, a *local rise of temperature in the right hypochondrium.* The temperature in this region may be as high as that of the axilla, or exceed it, while, under normal circumstances, it is two to five degrees lower than that of the axillary temperature. In the gastric crises of tabes such a local hyperthermia in the right hypochondrium is not observed.

In cholelithiasis the *liver* is either of normal size or enlarged, and this is due either to hyperemia, biliary stasis, abscess formation, or secondary carcinomatosis. In cases in which cholelithiasis occurs in cardiac patients, according to my experience a very frequent combination, the diagnosis of acute enlargement of the liver may cause perplexity, and the disease may either be attributed to gall-stone affection or be considered as acute stasis hyperemia. The pain may be due to the gall-stone or to tension of the capsule of the liver. I remember such a case of cholelithiasis in a patient suffering from mitral insufficiency in which at first I wavered in the

diagnosis and was in doubt as to the therapy to be instituted. Digitalis was without effect, but a carefully conducted treatment at Carlsbad caused the swelling and painfulness of the liver to disappear. The diagnosis of cholelithiasis was confirmed by the passage of small stones.

Enlargement of the liver, which is frequently observed in patients with gall-stones without jaundice, may be due to parenchymatous swelling or simultaneously active hyperemia of the organ, and this is, in fact, the view of French clinicians (*congestion hepatique*).

If to the cholecystitis calculosa an infectious cholangitis be added, the latter sufficiently explains the enlargement of the liver. There are, however, cases characterized by irregular fever, intermittent enlargement of the liver with sensitiveness upon pressure, yet without well-developed colic or jaundice. This rapid increase in the volume of the liver can hardly be explained except by the existence of acute hyperemia. This is illustrated by a case known to me in which, at the onset, a diagnosis was made of infiltrating carcinoma of the liver; but the rapid decrease in the size of the organ and the absence of pain scarcely permitted a different interpretation than that of cholelithiasis intrahepatica.

Diffused pain in the enlarged liver, the symptoms otherwise being pyemic, points to suppurative cholangitis, while a local sensitiveness to pressure outside the gall-bladder region, particularly when the pain radiates to the right shoulder, is found in abscess.

An enlargement of the liver with an *induration hard as wood,* chiefly in the region of the incisura, points to a carcinoma of the gall-bladder which has proliferated into the liver; particularly when cirrhosis is excluded and enlargement of the spleen is absent.

Elongation of the suspensory ligament in consequence of extreme dilatation of the gall-bladder, especially when hepatoptosis already exists, may produce a *wandering liver* showing extraordinary power of motion. *Perihepatitis* in cholelithiasis demands especial attention. In a case observed in the Clinic, a shoemaker, aged 53, had intermittent, irregular fever with chills, rises in temperature above 102.2° F. combined with colic-like pains in the liver, a conspicuously rapid pulse even in the afebrile and painless intervals, varying jaundice, with only occasionally bile-stained stools; distributed perihepatitis, manifesting itself over the whole extent of the liver by friction sounds, was the dominant feature of the clinical picture. The autopsy showed obstruction of the choledochus due to stone, dilatation of the upper portion of the same, of the hepatic duct, and of the bile ducts of the liver, ulceration of the wall of the common bile duct, and the formation of multiple abscesses from the size of a pea to the size of a bean, containing pus, mostly grayish yellow, but still bile-stained, and the walls of which consisted of discolored hepatic tissue. In regard to the auscultatory findings during life, such a general perihepatitis, if accompanied by other severe symptoms, must awaken suspicion that in the interior of

the liver a severe infection of the biliary passages has taken place with the formation of abscesses.

Compression of the hepatic artery may produce *aneurysm.* Such a case was observed by Schmidt. Here death occurred with the symptoms of fatal gastric and intestinal hemorrhage; the gall-bladder, filled with stones, communicated with the aneurysm.

The circumstance that aneurysms of the hepatic region sometimes produce murmurs reminds us again that *auscultation of this region* should not be neglected.

In examination of the liver the first point of attack is, naturally, the breeding place of gall-stones—*the gall-bladder.* If this organ, at the beginning of the attack, contracts about its fluid or solid contents, and provided the abdominal muscles are not too tense, it is found as a globular, sometimes fluctuating, tumor which may be palpated through the abdominal wall. Some patients themselves note the intermittent contractions of the gall-bladder, and in their history report this to be as though a tumor were being inflated in the right hypochondrium, but after cessation of the pain this disappears.

These sensations of the patients are very difficult to explain, as the intermittent swelling in the right hypochondrium may also be due to an accompanying acute enlargement of the liver. The contractions of the gall-bladder may be extraordinarily severe. In the case of a man who succumbed to reflex spasms during an attack, Gerhardt found impacted at the entrance of the cystic duct a large stone, splinters of which had broken off from the anterior end, and had penetrated the mucous membrane. The *gall-bladder tumor* moves upon inspiration, and is not fixed upon expiration, which is explained by its direct connection with the liver.

The *lateral, pendulum-like, displaceability of the tumor* is important in the differential diagnosis of tumors of the gall-bladder and of the kidney. In congenital elongation of the cystic duct the tumor may be very movable. Such a gall-bladder, displaced and filled with stones, and very painful, may be mistaken for wandering kidney.

In a case of Fournier's which occurred in a young woman, a diagnosis was made of myelitis, neuralgia, abscess of the liver and floating kidney, but, over the right kidney, an irregular yellowish tumor the size of an orange was found, which was formed by the gall-bladder filled with stones. The hypertrophic ductus choledochus and cystic duct had a length of from 15–25 cm., which explains the dislocation of the gall-bladder. But the tumor may also be immobile, and adhere to the convex surface of the liver, which, in certain cases, may lead to the assumption of hepatic echinococcus and abscess.

A tumor of the gall-bladder, which is distended in consequence of closure of the cystic duct, cannot, naturally, become smaller by manual compression. If, however, the obstruction in the cystic duct can be over-

come by this manipulation, a decrease in the size of the tumor may be brought about. A few instances of a gall-bladder of this kind, which may be expressed by paralysis or atrophy of the musculature, have been observed. Great distention of the gall-bladder may, in rare cases, produce permanent jaundice by compression of the hepatic or common gall-ducts. In lithogenous or cicatricial closure of the cystic duct the behavior of the gall-bladder depends, in the main, upon the texture of its mucous membrane. If the mucous membrane remains intact, secretion continues, and *dropsy of the gall-bladder* develops. In the opposite case, with marked damage of the mucous membrane the gall-bladder is obliterated, and becomes adherent to its surroundings. Stones may thus become encapsulated in the gall-bladder and form a harmless *caput mortuum,* as, for instance, an encapsulated bullet after a gun-shot wound. A gall-bladder of this kind filled with stones may sometimes be felt as a hard body in the incisura hepatis. Therefore, a lithogenous closure of the cystic duct is an important condition, especially for the surgeon, who most frequently deals with such adherent gall-bladders, less so for the internal clinician, who looks upon obliteration of the gall-bladder and encapsulation of the stones as a favorable conclusion of irregular cholelithiasis.

In lithogenous occlusion of the common gall-duct dilatation of the gall-bladder is very rarely found; usually it follows a preceding cholecystitis, while in carcinomatous compression or obstruction of the choledochus (excepting lithogenous choledochus cancer) distention of the gall-bladder is the rule. To this rule, which was established by Courvoisier, there are, however, many exceptions in either direction if we bear in mind that the condition of the gall-bladder depends, in the main, upon the extent of the mucous membrane lesion.

Impermeability of the cystic duct may be due to a lymph-gland lying alongside of it, which, according to Riedel, may be palpated at the operation as a small tumor and may be confounded with stones or cicatrices.

Acute perforation from pressure necrosis of the wall and ulcerative perforations of the gall-bladder sometimes leads to the formation of abscesses in the surroundings of the same, producing cavities, which are limited by the peritoneal, perihepatic, and pericystitic tissue formation. These cavities communicate with the gall-bladder, and may also perforate the abdominal wall, discharging greenish purulent fluid, and frequently stones, in large quantities. With an elongated gall-bladder, abscesses may thus form in the right iliac fossa. Such a deeply-situated, purulent cholecystitis may readily be confounded with perityphlitis, and, inversely, a vermiform process curving upward, or even adherent to the gall-bladder in the gall-bladder region, may resemble perityphlitis.

The tumors of the gall-bladder which are filled with stones may sometimes, by adhesion and displacement of the omentum, appear to be hard, and of considerable size.

A hard tumor in the gall-bladder region may be formed by a tongue-like process of the liver, if a corset lobe follows the direction of the slowly enlarging gall-bladder, and reaches beyond it. This so-called *Riedel's lobe* may give rise to difficulties in the differential diagnosis between cholelithiasis and syphilis of the liver. Echinococci bordering upon the portions of the liver in the incisura hepatis (original observation) may also lead to perplexity.

An excessive hardness of the distinctly enlarged and usually nodular gall-bladder, which is generally sensitive to pressure, and which then continues to the liver (*infiltration ligneuse* of the French), points to carcinoma of the gall-bladder, particularly when the infiltration of the gall-bladder is continuous to the liver. Hard nodules in the latter confirm the diagnosis. The lancinating character of the pains, meteorism, periodic vomiting, progressive jaundice and cachexia, a repugnance to meat and fatty foods, and monotonous anorexia which at the same time becomes prominent, which is permanent and invariable, enable us to differentiate typical cases of carcinoma of the gall-bladder from the lithiasis of old persons in which the appetite may be capricious, but in which, in the majority of cases, the painful exacerbations of the affection leave no doubt of its dependence upon gastric disturbance. Yet, even here, there are deviations from the ordinary type, as in a case observed in my Clinic.

Johanna A., a woman aged 56, was taken ill with burning pain in the gastric region and jaundice, and was treated by her family physician for gall-stones. During the entire course of the disease the patient had a good appetite, although it was somewhat decreased. Free HCl was determinable in the vomitus, pepsin was positive. Fifty per cent. of albumin solution was digested. Digestive leukocytosis present. The steadily growing tumor of the gall-bladder with increasing jaundice was shown at the autopsy to be due to an *endotheliosarcoma* of the gall-bladder.

While secondary stone formation in primary lithiasis of the gall-bladder is very frequent, we find in primary cancer of the gall-bladder, provided necrotic particles form the basis for stone formation and contraction of the gall-bladder is inhibited, that it is somewhat rare—therefore, in spite of biliary stasis and the presence of centers of crystallization, no gall-stones; in the rare instances of primary carcinoma of the gall-bladder, they did not consist of cholesterin but of calcium phosphate and carbonate.

In cases that are difficult of solution, in which the diagnosis between a tumor of the gall-bladder and one of the transverse colon must be made, naturally, all of the manipulations which are employed in the differential diagnosis of abdominal tumors, for example, inflation of the stomach and intestines with air and with water, must be employed. Upon bimanual examination in the left lateral position, and by making strong pressure against the right hypochondrium with the right hand, we sometimes suc-

ceed in pressing the gall-bladder to the right wall, and thus make it possible for the left hand to palpate the organ.

If the abdominal walls are flaccid, the gall-bladder is sometimes visible, which is, perhaps, impossible in the recumbent posture.

By adhesion of the stone-containing gall-bladder to the large omentum, the transverse colon may be lacerated, constipation, meteorism and the like may be the further consequences, and an atypical cholelithiasis may thus present itself under the picture of intestinal or pyloric stenosis.

By a contracted and displaced gall-bladder, and by pericholecystitic adhesions, the stomach, the duodenum, the gall-bladder, and the biliary passages may be torn; according to the tension of these organs, vomiting may be produced, and, by the bending of the cystic duct, intermittent colic.

From these adhesions between the stomach and biliary passages which resist all internal treatment, the *gastric symptoms* become prominent, and among the digestive disturbances which play such an important rôle in the picture of cholelithiasis *vomiting* is of special diagnostic import. Vomiting with severe retching, as a rule, accompanies the attack of colic, and generally produces little, if any, amelioration of the pain; while, in acute dyspepsia, gastralgia and ulcer, the pain usually ceases after vomiting. This vomiting in gall-stone affection usually occurs several hours after eating, but not invariably. In stone obstruction of the duodenal end of the common bile duct, irritation of the papilla of Vater may cause pain in the gastric region as well as vomiting soon after the ingestion of food, the condition simulating ulcer of the stomach or ulcer of the duodenum.

In addition, it must be emphasized that, according to Fleiner, in consequence of cholecystitis calculosa, actual gastric and intestinal ulcers may develop, as well as those of the large intestine, beginning from the serosa, which may perforate. Therefore, an accurate history, particularly in regard to the localization and radiation of the pain, should be obtained. It may be easily understood, however, and has already been observed, that true ulcer of the stomach, confirmed by operation, and particularly ulcer of the duodenum combined with jaundice and pains radiating toward the liver, has been mistaken for cholelithiasis.

Vomiting in gall-stones may occur in the early morning hours, as vomitus matutinus. A cook, aged 34, Rosalia Sch., who admitted the excessive use of alcohol, had attacks of gall-stone colic due to a tumor of the gall-bladder of the size of a walnut. Colic only occurred after midnight or toward the early morning hours, and ended with vomiting which appeared soon after arising from bed while the stomach was still empty. Frequently the origin of matutinal vomiting is very obscure. Although in the history of women suffering from gall-stones the abuse of alcohol is sometimes expressly emphasized, yet it is, nevertheless, a conspicuous fact that in classic atrophic alcoholic cirrhosis, according to my

observation, cholelithiasis neither shows itself as a complication during life, nor is it found at the autopsy to be auxiliary.

This experience all the more furnishes food for thought inasmuch as in cirrhosis, in spite of the profound damage of the liver-cells, the bile in the gall-bladder shows no conspicuous anomaly, at least, none in appearance.

Vomiting in gall-stone disease may be alimentary, bilious, stercobilious, or stercoral.

The gastric contents are usually normal or show diminished acidity; sometimes, however, there is even hyperchlorhydria, especially if the gall-bladder is adherent to the pylorus, secondary gastric dilatation, and irritation of the gastric mucous membrane by the retention of the ingesta.

Whether the gastric contents are vomited, or the bilious contents of the duodenum, depends mainly upon the intensity of the act of vomiting. As a rule, vomiting is so violent in gall-stone colic that the ejected material is bilious. In infrapapillary duodenal stenosis due to laceration and compression of the duodenum, the appearance of bile in the vomited material may be readily understood. On the other hand, in occlusion of the choledochus, the vomited material will contain no bile.

Uncontrollable vomiting with intense pain, great anxiety, constipation and meteorism, particularly in the hepatic region, is usual in perforation of the gall-bladder and in biliary abscesses. Uncontrollable vomiting of ingested food after the intake of nourishment, with severe pain in the hypochondrium radiating to the right shoulder and back, stubborn constipation, unquenchable thirst, and singultus were the clinical symptoms in a case of duodenal stenosis observed by Fauconneau-Dufresne which was due to compression of the tumor by the gall-bladder.

Stercobilious vomiting is almost always a sign of disturbance in the lumen of the intestine, and the appearance of stercobilin in the vomitus is of serious import in the course of cholelithiasis. I emphasize this because *intestinal occlusion* due to incarceration of stones in the intestine may, on the one hand, appear under the picture of *ileus* and run its course as the first manifestation of a latent cholelithiasis, or, on the other hand, gall-stones may appear under the mask of an acute intestinal stenosis, in which laparotomy reveals no obstruction of the intestine, neither invagination, torsion, nor stones, no peritonitis and no perforation; at the autopsy a large gall-stone is found in the common duct. These cases resemble those of nephrolithiasis, running their course under the picture of intestinal occlusion, of which I have seen 3 examples, and in this category also belongs the case of a fat woman, aged 58, who suffered from severe pain in the upper abdominal region, chills, fever and bilious vomiting. An irreducible umbilical hernia was found, painful upon pressure, which had, according to report, existed for some years. A diagnosis was accordingly made of incarcerated umbilical hernia. Laparotomy revealed cholecystitis

calculosa and nothing abnormal in the umbilical hernia. Removal of the stone was followed by recovery.

Large stones, which by perforation of the common duct or the gall-bladder reach the intestine, may produce symptoms of occlusion, and either soon after their entrance or a long time afterward, even weeks, months or years, severe symptoms of intestinal stenosis may appear. After prolonged retention in the intestine owing to peristaltic and antiperistaltic movements, they may become fixed in some portion by spastic contraction, either in the duodenum, jejunum or ileum, usually in the region of the ileo-cecal valve. Large stones may also reach the cecum and the large intestine after dilatation or ulceration of Bauhin's valve (ileo-cecal). In this manner even stones the size of a hen's egg may be discharged with the feces. In other cases, they lead to ulceration or perforation of the intestine with all their consequences. Rupture may occur directly into the peritoneal cavity and into cavities formed by preceding inflammation, and thus circumscribed abscesses develop. This shows how valuable for correct diagnosis are many obscure cases of intestinal occlusion or ulceration in the ileo-cecal region, and how important is an accurate history which may eventually point to a preceding cholelithiasis. This is all the more true since the stones which have reached the intestine by perforation may have been present for months and even for years in the intestinal mucous membrane without having caused distinct disturbance. For this reason close attention should be given to pains in the right hypochondrium and epigastrium, and to fever and vomiting occurring one or two hours after the principal meal and disappearing after a few hours. For the appearance of these symptoms after partaking of food may be the initial symptom of fistula of the gall-bladder and intestine, and, therefore, significant in the diagnosis of a succeeding stenosis of the intestine due to gall-stones. In cases in which the stones find their way into the intestinal canal, the cause of intestinal occlusion is not the mechanical closure of the intestine, for the stones are rarely so large as wholly to obstruct its lumen, and because, at the autopsy, the calculi are found to be freely movable in the intestine. The occlusion is often due to a contraction of the organ around the stone, which is directly proven by those cases of stenosis of the intestine due to gall-stone in which laparotomy is performed. In the case of Rogner-Gusenthal, upon removal of the stone the spasmodic contraction of the intestine around it was seen. Stones which are impacted in the duodenum simulate pyloric stenosis. Frequent vomiting, dilatation of the stomach, sensitiveness of the right hypochondrium upon pressure, contraction of the musculature of the abdomen upon the right side, or friction sound in the gall-bladder region, will indicate the correct diagnosis, as well as, usually, the absence of melena and hematemesis, and, under some circumstances, the results of chemical and bacteriological examination of the gastric contents. If the stones are impacted in the vicinity of the

ileo-cecal valve, appendicitis must be considered in the diagnosis. The localization of the pains in *McBurney's point,* rises in temperature, the age of the patient, etc., may lead to the true diagnosis. Stones in the sigmoid flexure have been mistaken for carcinoma. In very rare cases they may also lead to volvulus of the sigmoid. Diagnostically, it must be emphasized that in acute obstruction of the intestine from gall-stones the meteorism is usually slight; in volvulus of the sigmoid flexure, meteorism occurs early, increases rapidly, and reaches a high degree. In exceptional cases, the stones wandering in the intestine, if low down, and the abdominal walls flaccid, may be palpated by rectum in spite of meteorism and sensitiveness of the abdomen.

When the stone is lodged in the upper portion of the intestine, neoplasm is much more readily thought of than stone. In some cases of obstruction of the intestine due to gall-stones, the patients experience a sensation as though a firm body were moving in their intestines. Intestinal obstruction due to stone may be favored, that is, the stones may increase in size, by the apposition of fecal masses, such cases having been observed by Körte and Frerichs. Preceding colic, perhaps palpable tumor of the gall-bladder, the disappearance of the tumor with simultaneous symptoms of effusion of bile into the intestine, bilious vomiting, severe pains in the abdomen, and the symptoms of intestinal occlusion must all be considered in a diagnostic sense. The fact that stones have been previously found, the discharge of the same with the feces, as well as other symptoms of cholelithiasis, render the diagnosis positive. The occasional discharge of flatus, as well as the severity and rapid sequence of vomiting in intestinal obstruction due to gall-stone, in contrast to the longer intervals of vomiting in intestinal occlusion from other causes, may, in some cases, furnish a clue to the disease. The symptoms of occlusion may disappear and then recur. In the case of the aged a relapsing intestinal occlusion must suggest to us the possibility of a rupture of the stones into the intestine.

We see, therefore, that in the course of cholelithiasis the composition of the vomited material, the intensity of the act of vomiting, and the changes present in the lumen of the intestine may vary. First, as a rule, the contents of the stomach, subsequently those of the duodenum, and, finally, those of the jejunum and ileum are discharged. In the cases in which fecal vomiting occurs, the diagnosis of intestinal occlusion will not be difficult. If, however, the vomited material does not show fresh, unchanged bile, but bile which has already been acted upon by intestinal bacteria, which, therefore, originates from the lower portions of the intestine, its diagnostic significance as a premonitory sign of intestinal occlusion is obvious. In the early diagnosis of acute intestinal stenosis, the *vomiting of stercobilin* is, under some circumstances, an important finding, and I advise that an examination for stercobilin be made, particularly in

those cases in which, after laparotomy, the question must be decided whether the acute appearance of so-called bilious vomiting is the consequence of peritonitis or of a change in the lumen of the intestine.

Hematemesis occurring in long-existing jaundice may be a sign of the hemorrhagic diathesis, of ulcerative rupture of the biliary passages into the stomach, of secondary gastric ulceration, or the consequence of thrombosis of the portal vein.

That lithogenous carcinoma of the choledochus and of the duodenum may sometimes run its course with hematemesis and melena is obvious. This is the more apparent since cases have been reported in which large stones situated in the choledochus, with and without accompanying jaundice, the stones being palpable through the abdominal walls, have been confounded with carcinomata of the pylorus and of the duodenum. The absence of hydrochloric acid in the vomited material, and the signs of gastrectasis, may in such cases, as is reported in literature, assist in the diagnosis. Stones in the common duct may change their position upon respiration, and even upon palpation may show slight movability, as is the case in carcinoma of the pylorus, in which circumstance, however, they differ from carcinoma of the head of the pancreas.

With an existing jaundice the condition of the gall-bladder may be of diagnostic importance, the latter, as has been mentioned, in carcinoma of the head of the pancreas being frequently felt as a tensely filled tumor and also sometimes visible, while in a tumor of the gall-bladder stones in the common duct are frequently absent in consequence of a preceding contraction of the gall-bladder. The motility of stones in the choledochus, in contrast to carcinomatous obstructions therein, may also aid the surgeon during laparotomy.

Merkel describes a case of cholelithiasis which was very difficult to diagnosticate; it occurred in a woman, aged 51, who suffered from *distressing, uncontrollable singultus,* intense abdominal pains which were not localized, with exacerbations after meals, and who had a pear-shaped, painless tumor in the epigastrium. There was no jaundice, no vomiting; the stools were normal. The diagnosis wavered between tumor of the pylorus and movable kidney. Operation showed, after incision of the tumor which proved to be the adherent gall-bladder, a gall-stone weighing 15 grams, which was an exact model of the gall-bladder that had contracted completely around it. Recovery.

The differential diagnosis between gastric carcinoma and cholelithiasis may also give rise to perplexity in cases in which the gastric carcinoma is ushered in with the signs of an acute attack of cholelithiasis, yet without producing a palpable tumor.

Such a case I presented to the Clinic on Nov. 24, 1893; it occurred in a servant girl, aged 34, who reported that she had previously always been well, but was suddenly attacked by colic-like pains in the right hypo-

chondrium. These attacks steadily increased in frequency, and were followed by vomiting two or three hours after the ingestion of nourishment, loss of appetite, and, in the further course, jaundice. Free HCl was absent in the vomited material as well as lab ferment and lab zymogen; elongated lactic acid bacilli could not be demonstrated, but several eosinophilic cells were repeatedly found in the vomitus. Autopsy showed an infiltrated scirrhus of the pylorus with extreme stenosis of the latter, metastases of the mesenteric, retroperitoneal and periportal lymph-glands, compression of the common duct, and general jaundice.

In this case, which, on account of the sudden appearance of the symptoms, as well as by jaundice which was soon observed, resembled cholelithiasis, the *finding of eosinophilic cells in the vomited material* was of diagnostic importance, for the reason that these are significant of small cell infiltration of the interglandular tissue which consists of an accumulation of eosinophilic cells, as Hammerschlag demonstrated in gastric carcinoma.

The dilatation caused by cicatricial closure of the choledochus may reach an extreme degree, and may be palpated during life as a cyst-like tumor. Large stones impacted in the choledochus may compress the pancreatic duct and thus produce secondary infection of the pancreas and retention cysts; in complete closure of the panereatie duct, by preventing the panereatic juice from entering the intestine, they may give rise to extreme cachexia. The differential diagnosis of this latter form of cholelithiasis from careinoma of the head of the pancreas may, therefore, be exceedingly difficult.

Morgagni reports a case in which the common duct was dilated to the size of a stomach and filled with stones. Such enormous dilatations of the common duct may readily be mistaken for pancreatic cysts, all the more so as, on account of the topographico-anatomical relations of the common duct to the pancreas and also from clinical experience, secondary affections of the pancreas (closure of the pancreatic duct, indurative pancreatitis, retention cysts) have been proven to depend upon cholelithiasis. The diagnosis of these conditions becomes even more puzzling in view of the fact that intense pains having the character of gall-stone colic occur also in pancreatic cysts, as in a case which I showed in the Clinic upon June 16, 1901, and in which I believed myself justified in making a diagnosis of pancreatic cyst. A farmer, aged 67, from Galicia, reported that for twenty-seven years he had been suffering from pains in the right epigastrium radiating directly to the lumbar region, usually occurring at night and lasting from one to two hours. These pains recurred at intervals of from one to two months up to the year 1900. There was never any jaundice. Preceding the attacks the urine was said to have been sometimes remarkably clear; during the attacks it was of a reddish color, without sediment or blood, and without stone. The bowels during the

entire time were irregular and constipated, so that the patient took purgatives for many years. In August, 1900, the patient was again attacked by very severe spasmodic pains in the liver, this time accompanied by fever and sweating. There was vomiting and very marked meteorism; the pains were so intense that he writhed with agony. In February, 1901, a tumor slowly developed in the epigastrium. At present his abdomen and epigastric region are prominent on account of a globular tumor, the size of a head, which extends anteriorly from the right parasternal line to the left mammary line and reaches to the navel. The tumor shows a distinct vertical pulsation, synchronous with the heart's action, and distinct fluctuation. It is evidently displaced by respiration and pressure, and may also be moved laterally. Slight lateral displacement, particularly to the left, occurs when the patient lies upon the side. The tumor is almost painless. The feces are not excessive in amount, and are of normal appearance. Microscopically nothing abnormal is found in the feces or the urine. According to this history, a diagnosis of anything else than pancreatic cyst was scarcely possible.

Enlargement of the spleen does not belong to the picture of pure cholelithiasis, even when the attack runs its course with intense jaundice. Exceptions to this rule are formed by those cases in which infectious cholecystitis and cholangitis exist simultaneously; here the splenic tumor is of an infectious nature. In the diagnosis of secondary cirrhosis which develops in the course of cholelithiasis, enlargement of the spleen is significant, in contrast to carcinoma of the common duct and of the head of the pancreas, as well as of the glands at the porta hepatis which cause enlargement of the spleen only when they compress the portal vein. In metastatic carcinomatosis of the porta hepatis, which originates from cancer of the gall-bladder, enlargement of the spleen may be an infectious residuum of the precarcinomatous lithiasis of the gall-bladder.

An *examination of the feces* may give us valuable guidance in the diagnosis of cholelithiasis. In obstruction from stone in the common duct and in the hepatic duct, and in long-continued inspissation of bile, the feces, as is well known, become clay-colored, or ashy-gray. In lithogenous occlusion of the common duct, as has already been stated, the bile may occasionally flow into the intestine, provided stones which have become loosened move in a retrograde direction in the dilated common duct. This is in contrast to cicatricial and carcinomatous occlusion of the common duct, especially carcinomatous compression of the same, which is explained by the aggressive motion of the proliferating secondary mass. Tuberculosis of the glands at the porta hepatis may also lead to permanent occlusion of the common duct, but not invariably. I once observed a case in which the color of the feces varied, and, at the autopsy, stenosis of the choledochus was found, which was due to a mass of tuberculous glands. By an intermittent increase of the intrabiliary pressure, in consequence of the stasis

of bile above the stenosis, the variation in the color of the feces is explained similarly as the dribbling of urine in hypertrophy of the prostate gland. Analogous conditions may also occur in gall-stones which compress the common duct externally.

The patients sometimes first notice a whitish-gray color of the stools during the attack of colic, while they expressly emphasize that the urine a few days later resembles dark beer, and the skin becomes yellow. As regards the sequence of symptoms there are cases of gall-stone colic in which intestinal acholia precedes the jaundice and choluria.

Frequently this intermittent, ashy-gray color of the stools is combined with an acutely developing meteorism, which is the only objective manifestation of gall-stone colic.

A singer, aged 46, who was attacked by chills, pains radiating to the right shoulder, vomiting and diarrhea, reported that during the attack the abdomen became as tense as a drumhead, and the skin of the abdomen was glistening in appearance. The feces resembled those of an infant. There was no jaundice. This paradoxical phenomenon of *intestinal acholia without jaundice* requires special consideration, as it occurs particularly in those cases in which palpation of the gall-bladder reveals a distinct tumor. It is obvious in this instance that the gall-bladder acts as a reservoir, receiving the regurgitating bile from the over-filled biliary passages, which is naturally only possible when the cystic duct remains open.

The gall-bladder appears—at least in man and in many animals—to be a regulator of the intrabiliary pressure. This view is favored by the opinions of some authors who have observed a vicarious dilatation of the biliary passages after the closure of the gall-bladder and the cystic duct. Acholia of the feces without jaundice occurs also in fistula of the gall-bladder and abdominal walls after cholecystotomy; the explanation of this, according to Leichtenstern, may be that if a gall-bladder fistula exists biliary pressure cannot reach a degree necessary to overcome the tonus of the duodenal sphincter. Permanent intestinal acholia does not, therefore, justify the conclusion that an obstruction due to stone is present in the common duct, neither can it by any means be regarded as an indication for renewed operative interference.

According to Doyon, irritation of the central end of the vagus produces contraction of the gall-bladder and relaxation of the duodenal sphincter. It follows from this that a condition must exist in the living organism which necessitates the removal of this obstruction by antagonistic irritation of the peripheral branches of the pneumogastric nerve; therefore, this closed door may occasionally open, and become permeable for bile as well as for incarcerated stones in the common duct.

If, with the passage of stones into the intestine, the stasis of bile ceases, the retained bile pours into the intestine, and the acholic stools, or, at least, the stools deficient in bile, suddenly contain profuse amounts of

bile; the color of the feces in such cases depends principally upon the intensity of the intestinal peristalsis. If this is very intense, unaltered biliary coloring matter may be detected in the fluid stools. The feces are then yellow or green, and contain bilirubin and biliverdin. If the passage of the intestinal contents is so slow that the biliary coloring matter may be reduced, the feces become dark brown, sometimes tarry, and contain large amounts of stercobilin. The same is true in rupture of the gall-bladder or of the common duct into the duodenum. If, however, rupture occurs into the colon, it is evident that with the shorter road which the bile must traverse, and particularly with rapid peristalsis, the stools almost always contain undecomposed bile.

In the majority of cases the intestine is overflooded with accumulated bile, and *diarrhea* occurs. Ulcerating carcinomata of the biliary passages which perforate into the intestine may have the same effect. In a case in which, after the appearance of stools markedly stained with stercobilin without admixture of blood, and in which jaundice which had lasted a long time suddenly disappeared, the diagnosis of an ulcerative carcinoma of the common duct was made on account of the persisting marasmus of the patient. The autopsy showed an ulcerating villous cancer of the common duct. Accordingly, the composition of the stools may be utilized in the diagnosis of high or low perforation of the intestine.

Usually stubborn constipation is noted during the attack of colic. But there are also cases with diarrhea so profuse that, during the prevalence of a cholera epidemic, they may be mistaken for an attack of cholera. The diagnosis may also become difficult if poisoning, for example with arsenic, produce biliary colic; such cases have been observed. Stubborn diarrhea usually accompanies the forms of cholelithiasis which run their course in combination with acute septic infections, for example, endocarditis. But diarrhea, sometimes with hemorrhagic admixture, combined with anorexia, anemia and cachexia, may also be the principal manifestation of an insidious, but nevertheless severe, infection of the biliary passages.

Stones which have reached the intestine by perforation may, as is well known, be lodged in the intestinal mucous membrane for months, but they may also, as is shown by cases reported in literature, cause stubborn diarrhea. Such diarrhea may be due to ulceration of the intestine, particularly in the region of the ileo-cecal valve. In cases in which there is a suspicion of intestinal ulceration, it is advisable to examine the feces for pyogenic cocci, which, according to Gram, are positive (staphylococci and streptococci).

This aid to diagnosis, communicated to me by Kretz, has been successfully tested in our Clinic. As is well known, the majority of the normal intestinal flora of an adult consist of bacterium coli varieties, which are negative according to Gram, while cocci are present in the minority. In

ulcerative processes of the large intestine we find the pus flora positive to Gram, in contrast to intestinal flora negative to Gram, to be most prominent. The feces, dried upon a cover-glass, are stained according to Gram, and, as a contrast color for the microbes negative to Gram, a watery rubin fuchsin solution is utilized. In a case of tuberculous ulcers I found this reaction distinctly developed in the mucus particles of the stools, not, however, in the diarrheic dejecta of enteric fever, in which bacilli positive according to Gram prevailed in the microscopic picture.

Melena in cholelithiasis is produced by the same causes that give rise to hematemesis, which have already been enumerated. In rare cases of wandering stone from the irritation of the mucous membrane, the feces may show hemorrhagic admixture, and this must be especially borne in mind in the differential diagnosis of neoplasms or intussusceptions of the intestine.

Special weight must be attached to the *examination of the stone passed in the feces,* which is simultaneously a *corpus delicti* of cholelithiasis. Negative examination of the stool, however, does not permit the conclusion that no stones have been passed; for, according to Naunyn, stones of soft consistence may be disintegrated in the intestine. As we already know, the gall-bladder is the point of formation of stones. The stones which have wandered out of the gall-bladder, which have traversed the cystic duct, may, instead of taking their ordinary course into the wider common duct to the point of the duodenal papilla, move in a retrogressive direction, may be forced upward into the branches of the hepatic duct, and thus produce a cholelithiasis intrahepatica with all its dangerous tendency to biliary abscesses. This is the manner in which *cholelithiasis intrahepatica* of exogenous origin arises.

The development of stones in the branches of the hepatic duct may also be autochthonous in this locality, and without generating particular disturbance, they may find their way into the wider common duct. However, it is quite possible that the stones which have developed in the hepatic duct, instead of entering the common duct with the flow of bile, find their way to the gall-bladder where they then enlarge. We see, therefore, that stones in the choledochus may be of different origin, from the hepatic duct, from the gall-bladder, arising autochthonously by crystallization around cholesterin, and, finally, by the perforation of the gall-bladder and its diverticulum into the common duct.

Kehr has observed cases in which, in spite of large stones being impacted in the neck of the gall-bladder, small stones were found in the common duct. Stones may be situated in the cystic duct, and also simultaneously in the hepatic duct. After the extraction of stones from the gall-bladder, stones in the hepatic duct may move downward, which is of importance in considering the question of cholecystotomy or cholecystectomy.

The seat of the stone in some cases corresponds to a local area of

pain, which persists even when narcotics are administered to the patient, as, for example, injections of morphin.

The seat of the stone may sometimes be revealed by the sequence of the symptoms. In the overwhelming majority of cases preceding colic with local signs in the gall-bladder, a tumor, a collection of fluid, pain, then the disappearance of these symptoms, and an outbreak of permanent jaundice indicate that the seat of the stone is the common duct.

The *fate of the gall-stones in the intestine* varies. They may break up in their passage through the intestine, they may do much damage, small stones may be arrested in the appendix, and large ones in the ileo-cecal valve. Stones which have formed in the gall-bladder may, by producing a diverticulum, directly penetrate the substance of the liver, by perforation of the posterior wall of the gall-bladder they may produce a paranephritic abscess, after rupture they may appear in the lumbar region; in perforating downward they may also find their way into the urinary bladder, and, after long wanderings, we may be surprised to find them even in distant parts of the body; for instance, as in a case of Porges, who extracted a quantity of gall-stones from a fistula in the thigh of a woman.

In examining the stones passed in the feces, above all their size is to be noted. The question whether large stones can pass the conical, terminal portion of the ostium duodenale is variously answered by pathological anatomists. In opposition to the view of Rokitansky, who believes it possible, on account of the great power of dilatation of the common duct, that even stones the size of a hen's egg may pass the ostium duodenale choledochi, Roth maintains that such seeming dilatations are really typical perforations of the common duct, these being usually situated from 5 to 10 mm. above the ostium duodenale, and in these perforations the upper portion of the portio intestinalis ductus choledochi communicates with the descending part of the duodenum. The greatest dilatation of the ostium duodenale produced by gall-stones had, according to Roth, the diameter of a medium-sized pea. He does not believe it likely, therefore, that stones of greater dimensions reach the intestine naturally.

At the bedside, the passage of large stones after preceding grave symptoms is a sign of perforation, and with renewed colic forms, as Riedel says, a contra-indication to operation, as it is obviously possible that more stones may pass through the perforation. If at first small stones, and then somewhat larger ones pass, we are justified in concluding that the common gall-duct is dilated.

After a single passage of stones the biliary passages remain dilated, and this facilitates the later passage of succeeding stones so that, if small, they may easily be discharged in the feces.

The passage of equally large stones which are faceted may—*cæteris paribus*—form a contra-indication to operation, inasmuch as we assume

that the stones remaining in the gall-bladder may pass in the same way as did their predecessors. The repetition of attaeks without the passage of stones awakens the suspicion that large stones are also present in the gall-bladder. According to Riedel, the small stones which hide themselves in the common gall-duct, and occasionally slip into the hepatic duct, cause anxiety to the surgeon. A stone that possesses only one facet permits the conclusion that it is the first or the last of a series.

As the nucleus of stones, intestinal parasites, portions of the distoma hepaticum, and once the stone of a plum have been found.

Gall-stones are composed chiefly of cholesterin with a slight admixture of bilirubin calcium. Carbonate and phosphate stones must be regarded as pancreatic stones, but are not invariably so, as is proven by the two cases published by Eichhorst, who, in a case of mucous colic, found a profusion of small granular stones of from $\frac{1}{2}$ to 2 mm. in diameter which consisted of calcium carbonate. The seed of fruit (pears), the vertebra of birds, and chalk introduced for medical purposes must also be considered. It follows from this that the chemical, microscopical (for distoma), and perhaps also the bacteriologic (for typhoid bacilli) investigation of stones that have been passed may furnish valuable information regarding their origin, their past, and, eventually, also their etiology.

The amount of *urine* in cholelithiasis is either normal, diminished, or increased. A few cases have been observed of polyuria with bulimia and thirst but without glycosuria. In some instances, at the onset of the attack the excretion of a transparent urine in increased amount is reported (urina spastica). Retention of urine also occurs, particularly after the internal or subcutaneous administration of morphin. Choluria in jaundice is a well-known symptom. Conspicuous choluria without jaundice must lead us to suspect a perforation of the gall-bladder into the urinary bladder. Transitory albuminuria from hyperemia of the kidneys arises during the attack, and disappears after its cessation. Albuminuria with blood and casts occurs in cholangitis in consequence of infectious nephritis. Similar conditions are observed in occlusion of the common duct and chronic jaundice. Glycosuria, peptonuria, and acetonuria during the attack have been reported by different authors.

The excretion of sodium urate and uric acid as a *sedimentum lateritium* is not of rare occurrence.

In one case leucin and even tyrosin crystals were found for weeks. The excretion of indican is usually normal; sometimes, however, it has been found increased.

A woman, aged 38, the widow of a letter carrier, was admitted to the clinic with a diagnosis of cholelithiasis; during the attack, a conspicuous increase in indican was found. Operation revealed a contracted gall-bladder largely adherent to the omentum, which was tensely filled with stones. It may be that the adhesion of the gall-bladder to the omentum

and the consequent hindrance of peristalsis had an influence upon the increase of indican in the urine.

A marked increase of indican in the urine in cholelithiasis is evidently exceptional, in contrast to what is invariably found in perityphlitis, a circumstance which has been emphasized by Naunyn.

The doubtful value of the *indigo reaction* in the differential diagnosis is best illustrated by the two following cases.

In a robust baker, aged 55, in whom obstructive jaundice appeared with colic-like pains, constantly increasing anorexia, as well as diarrhea, the *constant absence of the indigo reaction* during his stay in the Clinic was particularly emphasized. Examination of the feces failed to show disease referable to the pancreas, nor was alimentary glycosuria present. With a probable diagnosis of "cholelithiasis with cicatricial closure of the common bile duct" the patient was transferred to the surgical division. There operation showed a *carcinoma arising from the head of the pancreas.*

A contrast to this was formed by the case of a book-keeper, aged 42, who was also transferred to the surgical division with a probable diagnosis of carcinoma of the head of the pancreas and occlusion of the pancreatic and common ducts; the daily examination of the massive feces, containing large amounts of fat and starch, revealed the constant absence of the indigo reaction, and justified to a certain extent the diagnosis of disease of the pancreas. At the operation (cholecystentero-anastomosis), the common bile duct was found to be of the thickness of a small finger, the duodenum with the large mesentery and the entire freely movable pancreas unchanged.

From many observations (Charcot, Regnard, Pick and my own) it is noted that the rises in temperature upon the day of attack in intermittent hepatic fever, contrasted with malarial paroxysms, correspond to a decrease in the amount of urea. According to F. Pick, this decrease affects not alone the urea, but also the entire nitrogen and ammonia excretion. An exact, quantitative urinary analysis, employing all the care which is necessary in metabolism, may also furnish valuable aid in those difficult cases in which the diagnosis hinges between hysteria and cholelithiasis.

In a patient who presented distinct symptoms of right-sided hemianesthetic hysteria I found, in contrast to the ordinary type of ovarian hyperesthesia, a marked sensitiveness in the gall-bladder region. Upon examination of the urine, it was evident to me that the precipitate which formed after the addition of barium chlorid and HCl was very voluminous, from which I inferred an increased excretion of sulphate in the urine; the patient had taken no preparations containing either Glauber's salt or sulphur. The pains in the region of the gall-bladder increased, and after three days a typical picture of hepatic colic with jaundice was presented. The qualitative analysis of a twenty-four-hour amount of urine voided during the attack revealed the following: Quantity of urine 1,040 c.c.,

urea 24.5, phosphorie anhydrid in eombination with earths 0.51, eombined with alkalies 2.09, urinary indigo but slightly inereased; biliary eoloring matter in small, bile acids in smaller, amounts; total sulphuric acid 6.22; combined sulphurie acid 0.19; relation 100 S : N = 47.83 instead of 15.1!

This was, therefore, a proof that the sulphurie acid did not owe its origin to the organie albumin rich in sulphur. Without endeavoring to explain this phenomenon, I should like to call attention to the excretion of sulphuric acid in the urine in cholelithiasis before the attack of eolie, particularly in febrile eases; here the excretion of nitrogen has been considered, but the relation of sulphuric acid to the nitrogen eurve has as yet been but little utilized for diagnostic purposes.

An exact quantitative investigation of the urine is, however, advisable for testing the function of the liver prior to operation. If points of support for the diagnosis of absenee of funetion of the liver eells may be gained from the urine (marked decrease of nitrogen excretion, espeeially glyeosuria and urobilinuria), the surgeon must observe a certain amount of reserve, because, in such cases, severe hemorrhage from the operative wound is to be expected just as in long-continued jaundice.

In *examining the blood* in cases of cholelithiasis with intermittent fever and chills, as has been stated by F. Pick, the leukocyte count may be of importance, in that the absenee of hyperleukocytosis in sueh cases is against suppurative processes in the liver.

In the Clinic of Prague, in Pick's ease of cholelithiasis with intermittent ehills and rises of temperature up to 104° F., in the eourse of which normal or subnormal leukocyte proportions were found in a number of attacks, he assumed an infeetion with the bacterium eoli. It may be seen from our observation that the cases of cholangitis due to infection with staphyloeocci, streptococci, and partieularly with diplococci, run their course with marked leukocytosis.

In the Clinic, in an attack of fever in a case of cholelithiasis with general infeetion by diplococci, a marked increase in the fibrin of the blood was found, besides leukoeytosis.

PROGNOSIS

In reconsidering what has been stated, it is manifest that *the outcome of cholelithiasis is, as a rule, favorable.* On the other hand, in the course of the disease conditions may arise which, in a comparatively brief time, cause death. In this category belong those cases of severe gall-stone colic in which death occurs from syncope, probably from abdominal shock, or in marantic individuals from cardiac weakness with degenerated heart muscle; furthermore, in this group belong those cases of acute cholecystitis whieh run their course with high fever and acute meteorism, becoming fatal either from peritonitis or general infection, from hepatic abseesses, ulcerative

and suppurative inflammation of the biliary passages and the portal vein, or empyema due to ordinary pus cocci, or ichorous empyemata with fetid pus in which, as in a case of Zuber and Leroboulet, anaërobic microbes were found. The dangers from obstruction of the common duct, cholemia, jaundice with hemorrhage appearing after a short time, and secondary acute hepatic atrophy, are no less threatening. The more chronic consequences and complications are biliary induration and cirrhosis, chronic suppuration of the peritoneum, adhesions with stenosis of the duodenum and of the remaining parts of the intestine by giving rise to gastro-intestinal disturbances, in some cases increased by morphinism, and, finally, cancer of the gall-bladder and biliary passages, all of which may cause death.

TREATMENT

On account of the many aspects of the disease and according to the stage in which we find it, the objects of treatment are manifold. At one time the colic most requires our therapy, in other cases closure of the cystic duct from stone, of the hepatic and common ducts, at other times the infection of the gall passages and peritoneal processes, or intestinal occlusion, and, finally, it must be our aim to prevent the formation of new gall-stones. In *the treatment of the paroxysms of pain,* opium and its preparations remain the sovereign remedy. Belladonna, atropin, chloroform and ether are advised for the same purpose. By some authorities the use of oil and glycerin is advocated. Hot poultices, hot cataplasms, in other cases the ice-bag, leeches, and protracted warm baths are the therapeutic measures with which to combat the pain.

In regard to *diet* there is still great diversity of opinion. According to experience, it appears that a mixed diet most thoroughly meets the indications. A plentiful intake of fluid, particularly the alkaline, chlorin-containing and saline mineral waters is favored by practice.

For the relief of the gastric and intestinal disturbances, as well as the jaundice, Fleiner has advised *gastric lavage,* which he believes to be more efficacious than mineral spring cures, but in regard to which I have no personal experience. Fleiner believes that by the influx of water into the small intestine, as occurs in gastric lavage, there is resorption of the same in the portal veins, and thus the liver and the entire body are washed out. Lately, systematic washings with silver nitrate solution at a temperature from 122° to 131° F. (50° to 55° C.) have been advised by Ehrlich.

These energetic procedures may, however, change a latent cholelithiasis into the actual stage of wandering of the stone. Stones which are latent, or which produce only slight difficulty, are to be treated as a *noli me tangere,* or, more correctly, "*quieta non movere.*" As the danger consists largely in the wandering of the stones, and this wandering is sometimes

induced by a Carlsbad cure, we must carefully consider whether it is advisable to send patients with slight symptoms to Carlsbad; in regard to the value of this old and popular remedy, a Carlsbad mineral-spring treatment, there is far from unanimity of opinion. According to Riedel, Carlsbad water alleviates the pain of cholecystitis by its purgative effect, by reducing the hyperemia of the inflamed mucous membrane of the intestine, but it simultaneously retards the expulsion of the stones. If the stones are passed while in Carlsbad, this does not occur because of, but in spite of, Carlsbad.

[In the treatment of cholelithiasis with infection of the biliary apparatus and in cholangitis without stones, many clinicians have found salicylic acid of value. Salicylic acid is excreted by the liver cells or at any rate, when ingested, it may be found in the bile. It is an antiseptic. It has been used sufficiently to prove its usefulness in infectious conditions of the biliary apparatus with and without gall-stones. It may be used alone or combined with alkalies and laxatives. I have used with good results the following:

℞ Sodii salicylatis..... 1.0
Sodii phosphatis Gran..... 2.0
Sodii sulphatis Exsic..... 6.0

Misce; signa. One teaspoonful in hot water one-half hour before meals, three or four times a day.

Or one may give phenol salicylas 0.3 in pill three or four times a day, and a sufficient dose of Carlsbad salts or magnesii sulphas in hot water on the fasting stomach to act as a brisk laxative.

Oleate of soda is also excreted by the liver and undoubtedly renders the bile more fluid. It may be given as a soda soap in the form of pills or in the form of ol. olivae in sufficient dose to act also as a laxative. Free intestinal action is of unquestioned value in cholelithiasis with infection. The "internal drainage" established by this method probably explains the frequent relief from cholangitis with gall-stones.

Gall-stone disease must be recognized as a surgical disease. The physician, with benefit, may utilize prophylactic measures, may reduce the accompanying *pain, vomiting* and *infection,* but only the surgeon surely may remove the calculus and correct the consequent morbid anatomy (adhesions, etc.). The danger of cholangitis, hepatic abscess, perigastric adhesions, pancreatitis, etc., occurring as a result of gall-stones is so great that even the most conservative physician may well hesitate to take the responsibility of non-surgical treatment.—Ed.]

The greatest difference of opinion prevails in regard to the question whether *early operation or conservative treatment* is indicated. In the cases in which the course is unusually protracted and painful and there is loss of strength, and in which repeated mineral spring treatments are

ineffectual, in those cases in which an empyema of the gall-bladder threatens perforation, or an irreducible stone obstruction of the common duct is present, no internal clinician will veto an operation. It is different, however, in regard to our justification for an operation as early as possible, and it would be interesting to know whether the surgeon who is convinced of the wisdom of radical operation, and who himself has gall-stones, would not prefer to give Carlsbad a trial before consenting to an operation. Certainly general rules cannot be laid down.

The method of operation is indicated after minute weighing of all the factors which are decisive for the topical diagnosis in a concrete case. Where the diagnosis has been incorrect, for example, when cholelithiasis has been diagnosticated as wandering kidney, and the surgeon, pursuing this incorrect diagnosis, attacks the supposed wandering kidney posteriorly, and finds a stone in the cystic duct, the uselessness of operative procedure in accordance with a laid-out program is apparent.

The specter fear of the serious consequences of cholelithiasis, which the surgeons love to oppose to a conservative treatment, is much weakened by contrary considerations. In the first place, quite a series of even desperate cases have been known which were nevertheless cured in consequence of perforation. Moreover, secondary stone formation in the tied stump of the cystic duct and in the biliary passages remains a sword of Damocles even after a successful operation, and, finally, in the cicatrix of operation a carcinoma may develop similarly as in the cicatrix of ulcer.

The surgery of the biliary passages is yet too recent to permit a conclusive opinion in regard to the *permanent results* of operation, and we are forced to agree with Wölfler, who says that surgery of the biliary passages has for several years presented no reliable statistics, and holds that those operated upon should be observed for at least a decade.

Since cholesterin stones, which alone come into question here, may develop in any part of the biliary passages in which there is mucous membrane epithelium, we can, in fact, speak of a radical prophylactic gall-stone operation only when it is possible to eradicate the entire cholesterin-forming mucous membrane epithelia.

ETIOLOGY

The treatment of cholelithiasis, particularly its prophylaxis, rests upon an uncertain foundation, principally for the reason that accurate knowledge of the near and remote causes of gall-stone formation is completely lacking. At present two theories chiefly dominate the *etiology* of gall-stones, the humoral and the infectious. The champions of the one regard cholelithiasis as a constitutional affection, a link in the chain of that diathesis whose family tree is arthritis. They base their opinions chiefly upon the occurrence of gall-stones in families in which, simultaneously,

obesity, gout, diabetes, hemorrhoids, eczema, increased uric acid excretion, and oxaluria, etc., are observed. The genesis of infection by means of the circulation or from the intestine is constantly gaining ground. This is based particularly upon the finding of the bacterium coli, and even upon the typhoid bacillus as the nucleus of gall-stones, as well as upon the similarity and the length of time the stones have been in the gall-bladder, which, according to Naunyn, may best be explained by a single bacterial infection.

In the humoral theory, it would be necessary to assume that the physiologic composition of the bile, that is, the relation between sodium-containing bile and cholesterin, has been altered in a pathological manner, and that the liver, to begin with, secretes a pathologically altered bile which readily forms a sediment, takes the shape of a ball, and grows into a clumpy stone. In the case of man, cholesterin is chiefly important since it forms the principal mass of gall-stones, while bilirubin calcium is to be looked upon only as a cement. The fact that cholesterin forms the chemical base of most human gall-stones is in contrast to the stones of the herbivora, which consist principally of bilirubin calcium, so that the gall-stones of oxen form a suitable material from which to prepare pure bilirubin in chemical laboratories. The influence of food and drinking-water, which is maintained by several authorities, would, therefore, be operative only in the case of bilirubin stones. Experienced cattle-raisers maintain that gall-stones are observed with surprising frequency in cattle and swine coming from regions where the meadows contain saltpeter, and this frequently permits a direct conclusion as to the regions from which the animals come. In the etiology of human stones, their rare occurrence with long-standing inspissated bile by the occlusion of the choledochus (catarrhal jaundice) is noteworthy, and the view is obvious that the causal factor of gall-stone formation is to be sought either in the blood from which the liver cells prepare bile, or in the liver cells themselves, which furnish a pathological secretion.

The chemical examination of the blood in cholesterin anemia, as well as in regard to its diminution of alkalinity and the deficiency of sodium-containing biliary acids, has been negative up to the present time. No investigations have as yet been made of hemoglobin in regard to its solubility, its power of crystallization, its faculty of coagulation, or its hygroscopic relation. It is possible that the hemoglobin varieties of different individuals are not identical, and that the hemoglobin of gall-stone patients is different from that of a normal person; such individuals may, perhaps, secrete a bile which crystallizes very readily, similarly as the patient with the urate or oxalate diathesis, who, without showing symptoms, intermittently excretes uric acid or calcium oxalate crystals in his urine.

If, however, to this congenital or acquired abnormality in the quanti-

tative composition of the bile, we add mechanical stasis, corset liver, obesity, pregnancy, large abdominal tumors, hepatoptosis, hindrance of the diaphragm action, senile atrophy of the musculature of the gall-bladder, a sedentary mode of life, infrequent meals, or bacterial infection, it is easy to conceive that, similarly as when uric acid stones are present, here also an agglomeration of cholesterin precipitate may result in gall-stones. In this sense, cholelithiasis may be regarded as a diathesis of the tissues.

By analogy, we might suppose that not the blood, but the liver cells themselves are affected, and that in their decomposition they furnish cholesterin, the possibility of which is admitted by Naunyn. In opposition to this is my personal experience, according to which gall-stones do not occur in alcoholic cirrhosis. It therefore follows that the destroyed liver cell, damaged by alcohol, furnishes bile which is not a suitable material for gall-stone formation although in its quantity, external appearance, color and consistence it does not decidedly deviate from the normal. Reports of microscopic and chemical examinations of bile in such cases are not at hand, and, so far as I know, have not been made.

Some of the factors are also in opposition to the theory of an infectious origin. In a general affection of the biliary passages the excretion of pathogenic bacteria is frequently observed. The presence of typhoid bacilli in the gall-bladder as the only bacterial flora—which makes an invasion by way of the intestine very unlikely, but which, on the other hand, favors the introduction by way of the circulation—is an ordinary finding in the course of enteric fever. Bacteriemia is frequent in ulcerative tuberculosis, while gall-stones in tuberculosis, as well as after a general bacterial infection, are seldom observed. Even in the cholelithiasis which follows these maladies an absolute relation between the diseases *cannot* be proven.

To explain acute serous, rapidly retrogressing cholecystitis, which develops a sterile, transparent dropsy of the gall-bladder by an infection from the bacterium coli in which the latter organism has been killed, appears to me somewhat far-fetched, if we take into consideration that infectious from the bacterium coli, as, for example, in cystitis or in pyelitis, are by no means of such short duration. Why should the gall-bladder in particular be so slightly injured by the bacterium coli, especially since it has been proven that bile is an excellent culture medium for this bacterium? In other mucous membranes we are familiar with acute swelling and increased secretion which cannot be regarded either as inflammation or as infection. I refer here particularly to coryza vasomotoria and to bronchial asthma, which cannot be regarded as infectious mucous membrane inflammations, either from a bacteriologic or clinical standpoint.

Some forms of cholelithiasis marked by the sudden disappearance of decided and painful tumors of the gall-bladder with all of their accompanying symptoms remind us of the turbulent catarrhal symptoms with

which bronchial asthma runs its course, a true "parturiunt montes" which, as Kehr remarked of infectious cholecystitis, "often resembles a fire of straw that blazes up but once and is rapidly extinguished." I presume that at least many forms of this gall-stone colic which have been explained as abortive infections belong to the group of *neuroses of secretion,* and are, therefore, in fact, *catarrhal asthma of the biliary passages.* For the gall-stone quiescent in the gall-bladder only occasionally assumes the rôle of a provocative agent for the development of colic, as, for example, nasal polypi in the development of bronchial asthma. Besides the abnormal secretion which is peculiar to asthma, spasm of the bronchial muscle is a prominent symptom; the accompanying fulminant nervous signs are analogous to those of gall-stone colic in which the secretion stored in the gall-bladder forms a motive power for the wandering stone. In acute serous dropsy of the gall-bladder, which at first is sterile but which by irritation of the foreign body and secondary bacterial infection may lead to cholecystitis, the effect of the operative removal of stones may be compared to that of the extraction of nasal polypi in asthma. Just as infectious bronchitis and lobular pneumonia may develop in the course of bronchial asthma, so secondary infections of the biliary passages play an important rôle in the pathology of cholelithiasis. Although it is evident that, in most cases, these infections merely change the latent to the actual stage of cholelithiasis, I am inclined to believe that quite a number of cases of gall-stone colic are neither inflammatory nor infectious. The capricious behavior of some cases, with a latency sometimes lasting for years, an eruptive appearance after psychical emotion and a sudden disappearance, the rapid sequence of relapses, the long intervals of health—these peculiarities are not readily explained by the assumption of a transitory infection, and therefore I regard these cases which run their course with acute dropsy of the gall-bladder as neuroses of secretion, parallel to those observed in catarrhal asthma.

Rokitansky[1] points out that pure cholesterin stones may be excreted without bile in consequence of the pathological secretion of transparent synovial-like fluid in dropsy of the gall-bladder, in complete occlusion of the cystic duct. This opinion was first clearly formulated by Naunyn who also found stones in a diverticulum, "surrounded by mucous membrane," which for a long time had precluded the presence of bile. Cholesterin is, however, produced by the mucous membrane itself, while bilirubin stones are formed only where bile is found.

These findings of Rokitansky and Naunyn alone favor the assumption that calculus formation is a local product of the epithelia of the gall-bladder, that is, of the epithelia of the biliary passages, and that the bile, as such, has no action in the original formation of the stones. Naunyn

[1] "Handbuch der speciellen pathologischen Anatomie," 1842–1844.

directly proved this latter fact by microscopic investigation; he showed that the original pulp of the gall-stone consists of structureless, soft clumps of cholesterin which project from the epithelium of the biliary passages and subsequently crystallize.

This is suggestive of the formation of Charcot's crystals which are the product of the crystallization of pre-formed eosinophilic cells; it is proven chiefly by the fact that the contents of pemphigus vesicles sometimes consist only of eosinophilic cells which on being subjected to a low temperature permit us to recognize the direct transformation of the decomposed cells into Charcot-Leyden crystals. The subject is an interesting one; in the surroundings of the parasite in the intestine eosinophilic cells form in large numbers, and by their subsequent decomposition furnish Charcot-Leyden crystals. The process appears to be similar in the mucous membrane of the biliary passages. The cholesterin nuclei which originate from decomposed epithelia are perhaps themselves the product of a pathologic secretion, analogous to that in bronchial asthma, and in their further course as foreign bodies they probably cause a local irritation which produces this pathologic secretion of the mucous membrane. In a wide sense a peculiar crystallizing catarrh is certainly present, but the etiology, the *primum movens,* of this catarrh is not bacterial infection. The latter may occur subsequently, and is often an undesirable complication. It is, however, not the cause, but the consequence, of stone formation.

It is possible that the entire epithelium of the biliary passages is not involved, but that only those layers which differ biologically and morphologically furnish the material for the excretion of cholesterin.

These considerations suggest fresh problems in the study of cholelithiasis, further examinations of the liver and bile, and particularly also of the blood in order to ascertain its power of crystallization and the coagulation of hemoglobin; they also show the importance of an exact microscopic examination of the secretion in cases of sterile dropsy of the gall-bladder, especially in those instances in which there is no infection from the bacterium coli, therefore, above all others, an examination of the fresh secretion of the gall-bladder in operative cases.

At the present time we are still under the dominating influence of the two theories that have been described, the humoral and the infectious, and although neither is in all respects satisfactory, we cannot be entirely independent of these so long as we possess no positive knowledge upon which to base other views of the etiology of gall-stones.

DISEASES OF THE INTESTINES

ACUTE DIFFUSE PERITONITIS, APPENDICITIS, AND PERITYPHLITIS

I. DIFFUSE AND CIRCUMSCRIBED PERITONITIS

By O. VIERORDT, Heidelberg

ACUTE DIFFUSE PERITONITIS

As an introduction to the discussion of our present views of acute peritonitis I will relate the following clinical history:

Case I.—A previously healthy merchant, aged 31, was taken ill after a few days of vague, dull pain in the right side of the abdomen which he had disregarded, and upon the 20th of October, about midday, he was seized with very severe pain in the right lower abdominal region which compelled him to seek his bed; soon afterward he had chilly sensations which increased to marked chills; there was also nausea, eructation and vomiting, first of food and then of bilious mucus; a little later tenesmus appeared, the patient first voiding small, compact feces, followed by scant, thin dejecta. Within a few hours the abdomen had become tympanitic, the pains continued with exacerbations upon motion, after eructations, and on talking; the entire abdomen was very sensitive. Strangury with the frequent discharge of scant urine was observed.

Toward evening the physician found the patient extremely ill, immovable in the active dorsal decubitus, with an anxious facial expression, reddened cheeks, cautious, superficial respiration with a low, hushed voice; he complained of continuous, also occasionally of marked tearing and contracting, pains in the entire abdomen, most severe upon the right side low down; the temperature was 103.2° F., the pulse was 112, full, somewhat tense, regular and even.

The lips were dry, the tongue markedly coated; *fætor ex ore* was present; painful eructations were frequent, also singultus, complete anorexia and extreme thirst. The respirations were superficial, quite rapid, and purely thoracic; the diaphragm was slightly raised; the pulmonary liver border was in the right mammillary line at the lower border of the fifth rib; upon anterior examination the thoracic organs appeared normal; the examination of the back was not then undertaken.

The entire abdomen was uniformly tympanitic, everywhere very sensitive to the slightest pressure, but more so upon the right side than upon the left. There was also pain upon pressure in the lumbar region.

Signs of abdominal respiration were absent. Careful palpation showed a uniform, drum-like resistance, otherwise nothing abnormal. The percussion note over the abdomen upon light tapping (and only this could be borne) revealed no decided difference, and nowhere any dulness; upon prolonged continued auscultation, high-pitched intestinal murmurs were here and there heard.

Retraction of the thighs produced diffuse abdominal pain, more marked upon the right side than upon the left; careful examination of the hernial rings gave a negative result.

Upon careful digital exploration per rectum in the dorsal decubitus, nothing abnormal was noted except pain in the floor of the pelvis; the rectum was empty.

Since morning neither feces nor flatus had been passed; the patient complained of strangury which, however, he rarely attempted to relieve because he feared to aggravate the pain which shot downward and radiated into the urethra. The urine was of high color, clear, and contained a trace of albumin and large amounts of indican.

The physician in charge of the case diagnosticated acute, diffuse peritonitis, the origin of which was not quite clear; very likely it was in the appendix. He ordered absolute rest, that the urine and feces be voided in the recumbent posture; that, for the present, only small quantities of ice be taken by the mouth; that two bags filled with ice be applied to the abdomen, and be suspended from a hook if they could not be borne directly upon the abdomen. Furthermore, at first every two hours, later somewhat less frequently, 0.03 of opium purum in powder form was to be taken in a little water.

In the following forty-eight hours, with irregular variations and a slight tendency to rise, the temperature ranged between 102.2° and 105.3° F. The pulse became more frequent but remained strong and uniform; the respirations were unaltered in character but increased in frequency to 48. The patient, unless under the influence of opium, was sleepless, his mind was clear, and he gave us the impression of being extremely ill, although not in collapse. The pains, eructation and vomiting were decidedly relieved by the opium; but ice-bags for a time were not well borne and cold Priessnitz compresses were substituted. Vomiting was rare, was invariably bilious and coarse-grained; neither feces nor flatus were discharged; the urine was as before, the diazo-reaction negative.

Distention of the abdomen and the area of diffuse resistance increased; sensitiveness to touch appeared to be dulled by the opium; in the ileo-cecal region, however, it was constantly severe and lancinating. The liver dulness below decreased; the pulmonary liver border extended to the upper border of the fifth rib; on the right side of the abdomen between the navel and the anterior superior spine of the ilium a circumscribed slight dulness was observed. There was great nausea and burning thirst.

Diagnosis: Acute diffuse appendicular peritonitis, probably also perforation; circumscribed perityphlitic abscess.

Operation was considered but not performed. Removal to the hospital for the purpose of an operation was absolutely declined by the patient.

I saw him upon the following day, the fourth of the disease. In general the severity of the clinical picture had increased, especially some of the individual symptoms: Severe, markedly febrile general condition; temperature about 104° F. and showing moderate remissions; pulse 120 to 136, moderately full, regular. There was insomnia with occasional opium slumber; otherwise the mind was clear but anxious. The tongue was thickly coated, the lips were dry, there was tormenting thirst. The cheeks were red. The patient maintained the dorsal decubitus with feebly flexed legs and hushed voice; the hands moved but slightly and trembled. Occasionally there were spontaneous attacks of severe, tearing, abdominal pain, starting posteriorly in the lower right side. The abdomen was very tympanitic and tense, and could scarcely be touched; nevertheless, it was possible to determine upon the right side low down an area of dulness about the size of a hand with increased resistance; otherwise the note was tympanitic upon percussion. The diaphragm

was raised; except for a small zone liver dulness was absent. Now and then there was grass-green vomitus which, the last time, contained a few brownish granules and had a fecal odor. Urine unchanged; micturition very painful; no feces.

Opium at first decidedly influenced the condition; the patient took daily 0.5 to 1.8, and since yesterday morphin subcutaneously 0.02 at a dose. Ice-bags were not well borne, and Priessnitz compresses were used continuously. The intake of food was reduced to almost nothing.

With a sharply circumscribed perityphlitic abscess there could be no doubt of the diagnosis of diffuse peritonitis nor of the indication for operation on account of the long continuance of the severe symptoms. But neither this proposition nor that of an exploratory laparotomy, the result of which might have induced the patient to yield, was accepted.

Morphin was ordered subcutaneously, Priessnitz compresses to the abdomen, pellets of ice and meat jelly by mouth; eventually gastric lavage.

Upon the sixth day of the disease the picture changed: The complexion became sallow, the face elongated, the eyes hollow; the pulse was 140, small, but quite regular; the temperature was 101.3° F.; there was clammy perspiration and a cool skin, the hands were cold; frequently slight eructations occurred and, now and then, ineffectual or mild paroxysms of vomiting of a greenish yellow material with a slight fecal odor. The mind was clear; there was little pain. The abdomen became somewhat softer, much less painful, and was readily palpated and percussed; there was a distinct resistance about the size of a hand, quite firm, and not fluctuating, and accompanied by marked dulness, around McBurney's point and downward, and only in this region severe stabbing pain; in other areas no dulness.

The symptoms were those of moderately severe *peritoneal collapse*; the prognosis was very grave although not positively hopeless. Treatment: Small quantities of alcohol, to be followed by camphor.

This condition lasted nearly twenty-four hours; then a very large and hard stool, followed by a thin one of hemorrhagico-purulent character was discharged and simultaneously a decided change took place: The appearance and pulse improved; the abdomen became softer with the exception of the marked resistance upon the right side low down, and the fever slightly remittent, its maximum 101.1° F. Vomiting did not recur; the patient moved about somewhat in bed and slept several hours in a half-lateral posture. Meat jelly and cold beef tea were swallowed.

Upon the next day there were several hemorrhagico-purulent stools, the urine was profuse and voided without pain. Nevertheless, a firm, flat resistance was still felt in the lower right side and upon pressure there was lancinating pain; no fever.

There could be no doubt that the perityphlitic abscess had ruptured into the intestine, and that in consequence of this the diffuse peritonitis had at once been relieved.

Treatment: Warm, followed by hot, flax-seed poultices; rest; freshly expressed meat juice or beef tea, in all 200 grams; thin gruel made with milk, 200 grams; wine, 100 grams in twenty-four hours, small portions to be taken every two hours; no drugs.

This improvement continued for several days and even became more marked. The abdomen returned to the norm with the exception of the ileo-cecal region; there was a small stool daily without recognizable pus; no fever.

Upon the *twelfth day of the disease* vomiting suddenly recurred, with severe, diffuse abdominal pain, marked meteorism, and fever to about 102.2° F.; the symptoms increased in severity, and changed during the night. The next morning I found the patient in collapse, his temperature 97.3° F., pulse 160, thready, uneven; conspicuous facies hippocratica; no pain; a slightly comatose condition, moderate

meteorism, no movement of the bowels. Stimulants were without effect; subcutaneous saline infusion revived the patient but only for a short time, and death occurred the following morning upon the fourteenth day of the disease.

Autopsy: Normal condition of the serosa above the omentum; the appendix surrounded by adhesions embedded in fecal pus, gangrenous toward its terminal portion, and showing perforation; fecal calculus in the pus; appendix movable toward the cecum. Agglutinated point of rupture at the median periphery of the cecum near the ileo-cecal valve. The perityphlitic pus appeared to be saeculated by adherent intestinal coils, but beyond the adhesions in the free abdominal cavity below the omentum there was diffuse, fresh, fibrinous peritonitis and distributed here and there small quantities of thin, putrid pus (many bacteria, large quantities of streptococci and coli bacilli). The peritoneum was injected, of a delicate rose-red color, here and there covered with fine, mucus-like pseudo-membranes. Heart flabby.

This clinical history is in every respect typical and instructive.

It shows us the origin of peritonitis which is by far the most common: in a diseased appendix. At the autopsy this was found necrotic and perforated. It is questionable whether the perforation existed from the onset of the disease; it is possible that at first an ulcer extending to the serosa caused an infection of the peritoneum; at all events this occurred acutely, and produced the sharply defined disease. The clinical abdominal symptoms in the first period of the malady pointed to the fact that at the onset there had been a diffuse inflammation of the peritoneum, and that later by the adhesions to the appendix which were found at the autopsy an early encapsulation of pus had taken place in the ileo-cecal region; this produced a purulent softening in the wall of the cecum and led to the favorable rupture of pus into the intestine and to an immediate amelioration of the acute peritonitis. The point of rupture, however, then closed, and partly to this circumstance, partly perhaps to the action of fresh infectious and toxic material, perhaps only to the perforation of the appendix, may be ascribed the exacerbation of the peritonitis, that is, a renewed attack which caused the death of the patient. In regard to the fulminant symptoms at the onset of the disease, however, it is more likely that even then perforation had already occurred, and that the final and fatal exacerbation was in consequence of adhesions formed in the first period which were powerless to resist the entrance of organisms producing inflammation. The pus finally broke through the adhesions, and produced diffuse peritonitis.

Moreover, the bacterial finding of streptococci and coli bacilli in the perityphlitic abscess is typical, and the limitation of the diffuse peritonitis to areas below the omentum is also instructive. This simultaneously prevented the invasion of organisms producing inflammation into the serous surfaces above.

This strong man, aged 31, had previously regarded himself as perfectly well. Nothing indicated the danger in which he found himself and which had existed since the appearance of the fecal calculus, the time

when this had formed being impossible to determine. The disease appeared acutely with fulminant symptoms.

Severe abdominal pain with tense abdominal walls, fever and vomiting form the characteristic triad in the first phase of the disease; less rapidly does meteorism appear. This depends upon the fact whether the inflammation of the serosa quickly spreads or remains local. Peritoneal meteorism is peculiar. The abdomen is uniformly distended, balloon-like; the muscles as well as the rest of the abdominal walls are tense. It must be added, however, that in spite of the excruciating pain upon touch there is no sign of contraction of the abdominal muscles, of the "muscular resistance" (*défense musculaire*) which is so common on pressure in other forms of abdominal pain, particularly when circumscribed. The same is true of the diaphragm; it is forced upward, the muscles are therefore elongated and tense; but there is no evidence of active contractions. Abdominal respiration ceases; gradually then, as may be recognized by the limits of percussion, increasing loss of muscle tonus is added. In this case the autopsy showed that the peritonitis had not advanced up to the serosa of the diaphragm.

The excessive abdominal pain, increased by movement and on the slightest pressure, caused the patient to remain motionless upon his back and to avoid the slightest movement of the abdomen either by speaking or coughing.

At the start the temperature was uniformly high, but later remissions in the pus fever were recognized. The pulse from the onset was comparatively frequent, regular, and somewhat tense.

The vomitus was at first composed of the gastric contents, then bile of a peculiarly pure, grass-green, biliverdin color mixed with a yellowish chyme-like material, and in the later stages of the disease showed thin masses having a fecal odor (ileus paralyticus). In regard to the dejecta, the two passages at the onset of the disease pointed to increased peristalsis; this was of short duration, soon changing to the opposite condition, and until the rupture of the perityphlitic abscess absolute constipation existed.

Among these signs pain, either spontaneous or upon touch, a rise in temperature, increased frequency of the pulse and, in general, the signs of severe illness, are to be looked upon as the local and general symptoms of a severe septic inflammation; vomiting, at least in the first stages of peritonitis, was due to decided reflex irritation of the numerous branches of the peritoneal nerves; the fecal discharges at the onset may be explained, but by no means invariably, as due to peristalsis acting reflexly. The constipation which followed this, however, as well as the meteorism, must be attributed to a hypotonia and paralysis of the musculature of the intestine by collateral edema.

Pain upon urination and strangury were due to inflammation of the peritoneal coat of the bladder, in which a noticeable irritation was pro-

duced by slight distention as well as by contraction of the bladder. The albuminuria was the well known infectio-toxic "febrile" form; indicanuria was in proportion to the fecal stasis.

In the course of the next few days a new symptom was added to this group: Exudation, which was demonstrable both by palpation and percussion. It was the natural consequence of inflammation of the peritoneum, and was both of diagnostic value as indicating general peritonitis and of special value in that, more definitely than the pain, it pointed to the original seat of the affection, which, according to present indications, could only have been an internal incarceration following right-sided inguinal hernia, or femoral hernia, or appendicitis. As neither the history nor the general status (normal condition of the hernial rings) furnished any points of support for the first view, only the diagnosis of appendicitis, that is, of perforation of the appendix, could be made with that degree of certainty attainable in diseases of the abdominal cavity in general.

After the appearance of these symptoms, a more or less firmly adherent but limited perityphlitic abscess, and a less intense although well developed peritonitis in this region, were assumed; the latter, notwithstanding the general painful meteorism, was not necessarily diffuse in the strict sense of the term; the omentum often protects the upper abdominal cavity from infection, as was proven in this case at the autopsy. It is possible that this diffuse peritonitis, which did not in the early period of the affection extend beyond the limited local focus, was not due to the intestinal contents and to bacteria, but chiefly to bacterial toxins which arose from the circumscribed original focus. This fact is pointed out by the prompt retrogression of the diffuse peritoneal symptoms after rupture of the abscess; the diffuse peritonitis of this first stage might then be designated a non-bacterial "*chemical*" inflammation, according to the terminology now in vogue; finally, it was positively a bacterial infection, although the post mortem finding of bacteria in the distant folds of the peritoneum is not proof of this; we know that during the terminal agony or after death these may wander a long distance from the perityphlitic focus.

After the symptoms of local and general inflammation with their secondary signs in the stomach and intestine had lasted for six days, suddenly a complete change took place: The nervous, anxious, extremely distressed patient became feeble and scarcely complained at all; his formerly congested face was pale and elongated, the nose pointed and cool; the skin lost its turgescence and warmth, and was covered with a cold sweat; the bodily temperature also fell, the pulse became small and frequent but remained quite regular, the abdomen became softer and to a great extent lost its sensitiveness; the vomiting decreased to a few painless attacks, and singultus disappeared: A picture which, to a certain extent, is a combination of collapse and narcosis although not to the degree of profound loss of consciousness, being the picture of an intoxication in

sharp contrast to the preceding febrile state. Just as the affection had suddenly developed to its full height at the onset of the disease, and much more swiftly than, for example, is the case in phlegmon of the external walls, so with extraordinary rapidity did the clinical picture assume a new type. In this respect we must consider the very great area of the peritoneal folds, their numerous lymphstomata, and their intimate relation to the circulation, and we are impressed with the fact that fluids and soluble, as well as formed, products are rapidly absorbed by the peritoneum.

Somewhat less rapidly than this, but nevertheless in the course of a few hours, another change took place, a favorable turn following the rupture of pus into the intestine. Here we were dealing with a well known and familiar phenomenon; if this occurs in the peritoneum the effects are particularly well marked; similarly as in the case of a phlegmon which rapidly disappears with the discharge of pus even although the inflammation extend beyond the pus focus, the symptoms of diffuse peritonitis promptly disappeared after the rupture. Very likely, as has already been stated, the symptoms of diffuse peritonitis in the first stages of the disease are to be referred to a chemical inflammation of the serosa, i. e., one due to toxins and without the ingress of bacteria; and it must be remembered that the clinical picture of this chemical peritonitis cannot be differentiated from that of the severe bacterial form. With the rupture of the abscess, the entrance of poisons into the free peritoneal cavity, and their resorption by the extensive peritoneal surfaces, as well as the vomiting and the intestinal paralysis, ceased. The taking of nourishment again became possible.

The point of rupture formed adhesions, the natural drainage of the peritoneal ichorous focus ceased, perhaps a new influx of inflammatory material from the perforated appendix also took place. There was a fresh relapse of the local peritonitis which extended beyond the boundaries of the limiting adhesions, and permitted the invasion by bacteria of the free abdominal cavity. This time the severe toxic picture of collapse immediately followed, and with marked decrease in cardiac strength led to death.

Doubtless the patient might have been saved in the first stages of the disease by the evacuation of the abscess; the incision would at first have acted similarly to spontaneous rupture into the intestine, but the relapse would have been prevented by permanent drainage, and a radical cure might have been brought about by the immediate or subsequent removal of the appendix.

Opium, no doubt, had a favorable effect upon the affection. By relieving intestinal irritability, and by bringing about a mild degree of narcosis, the patient was kept quiet and this materially assisted in limiting the severe perityphlitic suppuration in the first stage of the disease. If,

as it unfortunately happened, the point of rupture had not immediately closed again, if it had remained open until suppuration ceased and contraction and healing of the perforated appendix had taken place, opium would have been regarded as instrumental in saving the patient, and unquestionably, at least to some extent, justly so. Among other factors in the treatment, the relief to the intestine by the suspension of nourishment was of paramount importance. The subcutaneous saline infusions had an obvious but, naturally, only a transitory effect.

I have described this case in detail because it is calculated to exhibit to a great extent the present status, and our views, of the processes in acute peritonitis. The case occupies a middle ground between the very acute and the extremely gradual cases of perforating peritoneal inflammation; according to the valuable classification of Mikulicz it should be placed midway between an acute diffuse purulent and a progressive purulent peritonitis. The infection of the peritoneum at the onset was gradual, and not due to a gross perforation, for it may be assumed that the adhesions which paved the way for the favorable process in the severely infected region formed from the beginning, probably before the actual appearance of the affection, and therefore were unnoticed even by the patient himself.

The rapidity with which the peritoneum is invaded by the chemical bacterial infection and the general toxic action of the masses dominate to a preponderant degree the course of perforating peritonitis. In individual cases the virulence of the bacteria is naturally very different, and without doubt it has the greatest influence upon the intensity and extent of the inflammation, upon the quantity of toxins absorbed, and therefore upon the course of the affection; but in many cases it is probably only a secondary factor, for we know that the virulence of bacteria to a great degree depends upon the conditions under which they develop.

If, following this case which occupies a middle position, we consider some others which in an unfavorable as well as favorable sense more properly belong at the end of a series, the importance of this fact, that infectious processes in the peritoneum rapidly implicate its surroundings, will become still more evident.

I shall first quote a very characteristic case of Mikulicz's.[1]

Case II.—A man, between 25 and 30 years of age, was brought to the hospital in profound collapse; his speech was an idiom very difficult to comprehend, and only after great effort was it learned that for seven years he had suffered from an affection of the stomach, but had suddenly become very ill only a few hours before. His face was sunken, the extremities were cool, the pulse was 120 and moderately full. He repeatedly vomited masses having the odor of wine. The abdomen was uniformly distended and tense, painful upon palpation, with distinct signs of tympanites. A trial puncture by means of a fine cannula permitted the escape from

[1] *Volkmann's Vortr.* 1885, Nr. 262.

the abdominal cavity of gas which was inflammable and had the odor of alcohol; after the puncture subcutaneous emphysema occurred in the surroundings of the opening.

According to all the signs, a diagnosis of perforating peritonitis was hardly questionable, but the origin was not clear.

The same evening laparotomy was performed. Incision was made in the median line from the navel to the symphysis; upon opening the peritoneum gas and a great quantity of fluid of a coffee-brown color and with the odor of wine, also undigested rice granules, escaped. The entire abdominal cavity was filled with this fluid; the intestine, except for isolated superficial ecchymoses, was of normal appearance. Fluid oozed from the gastric region, and, after enlarging the incision, a slit-like opening 6 to 8 cm. in extent was found in the lesser curvature of the enormously dilated stomach near the cardia.

The stomach was emptied, the opening closed, the abdominal cavity washed with thymol, and the intestines cleansed.

Collapse followed the operation and, in spite of stimulation, death occurred three hours later.

At the *autopsy* a greatly distended and flabby stomach was found, with normal mucous membrane borders around the previously mentioned slit-like opening which had been sewed up; no ulcer; in the vicinity of the posterior upper angle of the wound a striated cicatrix about the size of a ten cent piece, and having no connection with the wound.

The peritoneum everywhere was smooth.

Here, in the course of a few hours, the *severe picture of collapse* developed, resembling that of our first case, with positive local signs of diffuse peritonitis. The operation, which was certainly indicated, may have slightly hastened the end, but, even without this, life would have terminated in the course of a few hours. Rupture of a dilated stomach in the neighborhood of an old cicatrix, undoubtedly due to a former ulcer, was found. There was no gradual inflammation continuing from the serosa, but, on the contrary, a sudden rupture into a previously perfectly normal peritoneum, perhaps due to the cicatrix of an ulcer, with immediate and overwhelming flooding of the peritoneal cavity with the gastric contents.

In rapid sequence was a painful distention of the abdomen with accompanying fulminant symptoms which clinically resembled peritonitis and which, if life had continued, might without doubt have been considered the precursors of this affection, also an immediate and very grave general condition which the earlier physicians designated as "reflex" or "nervous" shock, and the newer school is inclined to attribute to a "septic" intoxication; to this a third fundamental condition was probably added: Congestion of the vessels in the splanchnic area in consequence of congestive hyperemia of this vascular region.

The designation, "perforative peritonitis," is naturally unsuited to a case of this kind, for the patient died before the appearance of inflammation, consequently, not as its result; the most recent designation, "peritoneal sepsis," according to what has been above stated, is, in our opinion, too one-sided. We are evidently dealing with a most unusual case of

accidental perforation into the free peritoneal cavity, with masses of deleterious products distributed over the surface of the peritoneum which is capable of such enormous absorption, these products being the probable result of a purely chemical and local effect, as in the circulation, for there had scarcely been time for mieroörganisms to develop to any extent.

It may appear remarkable that, in such a case, the cleansing of the abdominal cavity and the closure of the death-producing rent should offer no chance for improvement. But the operation, without doubt, adds to the first shock another, it produces a fresh congestion of the abdomen with increased internal hyperemia, and even to-day, when, as far as these dangers are concerned, laparotomies are performed with every possible precaution, similar cases constantly demonstrate that it is frequently impossible to bring about a favorable result.

In contrast to this case I shall give briefly the notes of a perforation of the appendix running an acute course, and dating from a previous attack in which less favorable results were obtained by operation:

Case III.—A boy, aged 13, previously well, was taken ill late at night after a cold bath with severe abdominal pain upon the right side anteriorly and low down and with fulminant vomiting; the abdomen began to swell and there was no fecal movement. The physician called during the night ordered opium and ice.

The next morning the boy was admitted to the hospital: The patient was pale, the mind clear; the temperature was 100.9° F., the pulse 120, very small and regular; there was considerable abdominal pain, particularly on the right side, low down. The abdomen showed a uniform, drum-like swelling, which was extremely sensitive and most marked in the ileo-cecal region; no tumor or dulness, hernial rings free, no bowel movement. The urine was scant, caused pain when voided, was very dark in color, and contained much indican.

Diagnosis: Perforation of the appendix. *Treatment:* Opium, ice locally, pellets of ice by month, and camphor.

In the course of the afternoon the patient frequently vomited small quantities of grass-green material; collapse appeared; the bodily temperature fell to normal, the pulse was 140. Respiration became difficult, the diaphragm rose very high, the abdomen became still more swollen, and was now enormously distended.

During the night the patient became unconscious (opium was discontinued). Toward morning the mind was again clear; there were a few attacks of vomiting, but no longer any complaints; the abdomen again became soft, the face still more sallow, the pulse imperceptible. Now and then delirium with hallucinations was present. Death occurred upon the third day of the disease.

Autopsy: Gangrene and perforation of the appendix; fecal calculus. Fresh diffuse peritonitis with scant, turbid, serous exudate and the finest fibrinous deposits. No sign of adhesions about the cecum.

Here the conditions are similar to those of the preceding observation of Mikulicz's; the perforation was into the free abdominal cavity, at once produced severe pain and fulminant vomiting, and, by the rapid distribution of putrid masses upon the serosa, early symptoms of peritoneal collapse which within forty hours caused death. The autopsy showed peritonitis to be present, but only in its first stages.

Proceeding from these cases we next meet with those in which well developed peritonitis takes place, but in which a limitation to the immediate surroundings of the point of origin is out of the question. A classical example of this form of acute diffuse peritonitis is the following:

Case IV.—A saleswoman, aged 21, previously healthy, a primipara, was attacked upon the second day after a quite severe but non-instrumental labor (small laceration of the perineum, decided hemorrhage ex utero) with a chill, vomiting, and moderately severe, diffused, abdominal pain.

Upon the evening of this day the patient was very ill, the mind was clear but showed excitement; the temperature was 105.4°, the pulse 120, the respirations frequent and somewhat labored; there was repeated and distressing greenish vomiting, also singultus. The diaphragm was very high, the abdomen greatly distended, balloon-like, and very sensitive. Careful percussion elicited a tympanitic note throughout, no liver dulness; some few splashing intestinal murmurs were observed; during the examination diarrhea with tenesmus-like pain. Hernial rings were free, nothing abnormal was observed about the genitalia, and the same was true of the discharges. Much indican and some albumin were present in the urine.

During the night collapse occurred with cold sweat and a temperature of 97°; the pulse was 140 and very small. The patient did not rally from this collapse: At the beginning she was still very excited and delirious, but gradually became more quiet; most of the time her mind was clear. There was pallor, the internal temperature gradually rose to 101.8°, but the extremities remained cool; the abdomen became softer and less sensitive. About thirty-six hours after the onset of the attack deep percussion of the lower lateral region on both sides showed small areas of dulness. The uterus was moderately contracted and very sensitive.

The dyspnea increased; during the second night the patient was periodically restless, threw herself about, was somewhat delirious; she vomited with effort and in paroxysms, particularly when fluid was administered, ejecting greenish-yellow masses; two thin, scant, friable bowel discharges were evacuated in the bed. The patient had no pain, lucid intervals alternated with delirium; there was extreme pallor, facies hippocratica; dyspnea was slight, the abdomen was quite soft, the pulse scarcely perceptible. In the succeeding night death resulted from cardiac weakness after a duration of the illness for not quite three days.

Autopsy revealed a diffuse infra-omental peritonitis with moderately profuse thin pus and slight adhesions of some intestinal coils. In the corpus and collum uteri numerous pus foci, some of which extended to the peritoneal coat. No pyemic foci in other organs. No bacteriologic investigation was made.

In this case pus foci in the wall of the infected puerperal uterus produced a very acute peritonitis with peculiar signs of intoxication in that these foci spread to the peritoneum without producing extensive perforation, a condition which we partially recognized in the first two observations. Here we are dealing with a typical picture of *peritoneal sepsis*. The peritoneal symptoms were clear, they made an early diagnosis possible; there was no doubt as to their origin. It was either in the uterus or in its adnexa. A striking feature of the pathologic picture was the early collapse which was probably in part to be attributed to labor which had just taken place. The diarrhea in this case was conspicuous, and this is a

characteristic of puerperal peritonitis to which various authors, and recently Nothnagel, have called attention.

At the time in which this case occurred, and for such cases, surgical treatment was seldom resorted to; in the case in question it could have been of little avail. Internal treatment was entirely without effect.

These peritoneal inflammations which start from the point of invasion and rapidly implicate large areas of the peritoneum are in contrast to other forms in which there is a more or less conspicuous tendency to limitation. I shall first quote a classical case reported by Mikulicz.[1]

Case V.—A school-boy, aged 15, was attacked without prodromes by severe abdominal pain and vomiting; in the next few days the abdomen became distended and there was moderate fever. A fecal evacuation occurred daily.

Upon the fifth day: Pulse 100, quite strong, temperature somewhat elevated. Abdomen uniformly distended and everywhere sensitive to pressure, particularly in the ileo-cecal region, where there was increased resistance and limited dulness extending from two to four fingerbreadths from the crest of the ilium and reaching up to the lumbar region.

Incision in this region was followed by the discharge of 200 c.c. of thin fluid pus from a cavity limited by loosely adherent intestinal coils and omentum. The cavity was packed with iodoform gauze and opium and moist heat were ordered.

No noteworthy improvement followed this, but the dulness increased; there was bilious vomiting, no flatus. For this reason upon the following day, the seventh day of the disease, the first incision was extended downward and toward the median line. Ichorous pus exuded from every area limited by fibrino-purulent adhesions of the appendix, several coils of the small intestine and the omentum; the appendix, which was 8 cm. in length, showed a perforation at its middle and adjacent to this a fecal calculus. The operation consisted of resection of the appendix, intestinal suture, careful cleansing (boric acid solution), and packing with iodoform gauze.

The meteorism and the sensitiveness slowly decreased; flatus and later a fecal movement were discharged, but vomiting still persisted. The temperature remained at 100.4°; the pulse was somewhat lower, 104 to 116.

This improvement continued for six days and up to the twelfth day of the disease, when, with a slight rise in temperature and increase in pulse rate, a new area of dulness with resistance and pain upon pressure appeared over Poupart's ligament on the left.

Upon the fifteenth day of the disease another incision was made: A half liter of foul-smelling pus was evacuated from a cavity between adherent intestinal coils which extended into the false pelvis. The cavity was irrigated and drained, and improvement followed.

Upon the nineteenth and also upon the thirty-third day of the disease, accompanied by mild febrile symptoms, two new exudates appeared, the first anteriorly in the median line between the navel and the symphysis, the second upon the right in the true pelvis, and this could be reached from the opening of the second incision. Both were incised and discharged pus. Drainage.

Later still there was a small pleural exudate upon the right side which healed spontaneously, the patient leaving his bed after three months; there was still a fine intestinal fistula originating from the stump of the appendix; it soon closed. A few attacks of colic occurred, then convalescence and restoration to health followed.

[1] *Mikulicz*, "Ueber die operative Behandlung der Perforationsperitonitis." *Verh. der deutschen Gesellschaft für Chirurgie*, 1889.

This is the case which, if I am not mistaken, Mikuliez denominated as a so-called *progressive purulent form of perforating peritonitis.* Here we may assume that, prior to the manifestations of the affection, toxins or isolated bacteria entered the wall of the already changed appendix and produced a mild plastic peritonitis encircling it. The rupture of the appendix, with which the obvious symptoms appeared, occurred in a region preformed by adhesions; the adhesions, however, were not close enough for chemical poisons, perhaps were not even sufficiently invulnerable to microbes; this is proven by the immediate symptoms of general peritonitis; we have here the typical picture of attenuated general peritonitis (either chemical or bacterial) in the area surrounding a focus of intense inflammation not sufficiently limited by adhesions. We may consider that this diffuse attenuated inflammation was due to toxins which constantly traversed the actual focus composed of pseudo-membranes, and that later bacteria also now and then penetrated this wall and produced the various fresh suppurations some of which extended to distant regions of the abdominal cavity. It is also possible that bacteria were present from the onset, and gradually, in isolated areas, found especially favorable soil for their growth. At all events there was but a moderate local and general toxic effect, and this is obvious from the fact that with slight interruptions feces and flatus were discharged, that the fever was not high, and that not the slightest sign of peritoneal sepsis was ever observed.

The peculiar features of plastic inflammation, which in itself produces resistance, but which may, under some circumstances, be again overcome by the toxins of the disease, we shall meet again in a more distinct form and running a much slower course in the description of tubercular peritonitis. In this case everything occurred in very rapid tempo. Anatomically it is especially interesting to note the important rôle which the omentum plays in limiting inflammations in the abdomen.

As an aid to the diagnosis continuous, persistent, deep palpation (also per rectum!) is very valuable, as is also deep percussion of the entire abdomen in the prompt recognition of pus foci. The focal treatment of the affection is very interesting; the radical procedure of loosening the adhesions at the onset, with the toilet of the entire abdominal cavity, could hardly in this case have produced such brilliant results.

This case, in fact, proved to be conspicuously "progressive." This progression may also take place after apparent consolidation of a local perityphlitis, and with such rapidity that a sudden rupture of the pus focus is simulated.

An observation of Sonnenburg's is a characteristic illustration of such a course:

Case VI.—An apprentice, aged 16, previously well, was attacked with severe pain and vomiting. Upon the third day he was quite ill. The temperature was 101.7°, there was congestion with increased respiration, and a full pulse of 100.

Upon the fourth day an area of resistance about the size of a plate with dulness below the right arch of the ribs amounting to two fingerbreadths and moderately sensitive to pressure was observed; the rest of the abdomen was soft and only slightly sensitive; examination per rectum negative; urine only upon catheterization. The temperature had fallen to 100.8° and, after a careful emptying of the rectum by means of a glycerin suppository, gradually fell to 99.3°; simultaneously the tenderness of the abdomen entirely disappeared but the resistance and dulness upon the right side remained unchanged; the pulse was normal, the appetite better, the subjective condition excellent. Upon the following night and the evening of the sixth day of the disease the picture of severe diffuse acute peritonitis suddenly developed with rapidly rising temperature, a pulse of 140, and great restlessness; the next morning the patient was in collapse, the entire abdomen was sensitive, dulness was distributed; at midday the temperature and pulse fell, the abdomen became softer; during the night the condition became worse and death occurred early upon the eighth day of the disease.

Sonnenburg assumes in this patient, who declined operation, the rupture of a perityphlitic abscess into the free abdominal cavity, and he is probably correct.

The patient was not operated upon in the first stage of the disease, the time chosen in by far the majority of all cases of perforating peritonitis which from the onset show a definite localization, in which the rest of the abdomen is not implicated, and in which the general symptoms steadily improve. A further limitation and a transition into a relatively non-irritating chronic stage or even a cure was expected. That, however, we cannot count upon this with certainty is demonstrated by this case; and it proves how difficult it is to prognosticate the course of such an affection. Such cases are calculated decidedly to bias the opinion of those who see them as to the advisability of operative interference.

Fortunately, however, such cases are infrequent.

In comparison I shall relate the brief clinical history of a case of moderate circumscribed perityphlitis, and one that remained such. This closes the series of the forms of peritonitis arising from inflamed neighboring organs, the series beginning with the severest cases and ranging to the mildest.

Case VII.—H. G., a robust student, aged 22, in the last five years suffered three times from "appendicular inflammation" of brief duration, the last time, according to report, accompanied by a chill and high fever. He was seized in the evening after a fatiguing bicycle ride with a chill, followed by fever, repeated vomiting, and severe pain in the right side of the abdomen; the next morning the temperature was 103.2°, the pulse 120, the general condition moderately severe, violent pain; he vomited bile, there was no fecal movement.

The tongue was thickly coated; between the navel and the spine of the ilium an enormously sensitive area of resistance about the size of a small apple was found. The surrounding parts were also sensitive to pressure as well as the posterior lumbar region. The hernial rings were free; no bladder symptoms. The rest of the abdomen was soft and not sensitive to pressure. The treatment was by ice, small doses of opium; no food allowed.

Upon the second and third days of the disease, while the vomiting had ceased, the temperature remained between 100.4° and 102.2°, the tumor had enlarged to the size of a small plate, was flat, coarse, very sensitive, immovable, with a dull note upon percussion; no fluctuation. No fecal movement, great thirst. The rest of the abdomen was not involved. The patient was quite ill and in severe pain. Moderate amounts of opium were ordered.

Early upon the fourth day, the temperature was 100.2°, in the evening 101.1°; there was no further enlargement. In the evening a compact, dark-brown stool was voided, and in the night another, of pappy consistence, which was not seen. The pain was slight; the temperature had fallen to normal; a general improvement in the condition was noted.

From this time there was no fever, the bowels moved daily without recognizable pus or blood, and the tumor steadily decreased in size; hunger reappeared, and within eight days, except for a deep-seated indistinct resistance over McBurney's point, all the symptoms had yielded. This resistance was demonstrable for some time; then it, too, disappeared.

It had been my intention to propose an operation after the attack abated, but I relinquished this plan in consequence of the complete disappearance of the symptoms.

From this time the patient continued perfectly well for six years.

This case belongs to the extremely full category of moderately severe cases of limited perityphlitis. Perforation is here most unlikely. The condition is probably due to chronic, paroxysmally exacerbating attacks of appendicitis without fecal calculus, but perhaps a slight stricture. In the last attack (whether in the former is questionable) a purulent periappendicitis surrounded the isolated appendix to which adhesions had long since formed, and, according to the position of the tumor, probably also the medial side of the cecum. The quite fulminant discharge of pus in the following days did not rupture the barrier, but only strengthened it. There was no fluctuation. The change in the condition was probably due to a rupture into the colon which rapidly drained the abscess, and in consequence healing soon took place. Subsequently the fresh inflammatory formation was absorbed, and the appendix—as for six years nothing abnormal could be felt, there was no sensitiveness, and no relapse occurred—was probably obliterated.

A favorable course was looked for, although it could not be *prognosticated with certainty* because two milder attacks had preceded this, the disease setting in with moderately fulminant symptoms, without implication of the rest of the abdomen, and without any signs of peritoneal collapse, and because a tumor at once appeared which was very coarse and slowly increased in size. The probability of a favorable course became still greater upon the fourth day when it was observed that the tumor no longer increased in size and the fever declined; but even then the prognosis could not be regarded as quite certain, and the disappearance of the fever and the return of a feeling of well-being did not remove our anxious fear of a relapse, possibly of a still more serious character. This, however, was, according to general clinical experience, to a high degree unlikely.

I am strongly tempted to present as complete a picture as possible of the numerous varieties of peritonitis by quoting still other pathologic pictures. This temptation must be resisted for it would lead us too far from our subject; to be exhaustive in any sense it would be necessary to cite so many isolated cases that the result would be confusion instead of clearness.

Neither is it my object to present a description uniformly elaborated in all directions like a text-book description. On the contrary, when referring to previous histories, points will be described which are regarded as essential to the etiology and to the clinical picture, and which will chiefly aid us in deciding upon the treatment that is to-day recognized as fundamental.

In clinical histories we have learned to recognize inflammations of the appendix, gastric and intestinal ulcers, and septic infections of the uterus as causes of peritonitis; these are partly caused by perforations, partly by propagation through the lymph-vessels. We must still mention other frequent sources of peritonitis: The biliary passages whose sterile contents cause little or no chronic inflammatory irritation, but which, when infected and according to the virulence of the infectious agent (mostly bacterium coli) may cause the severest acute peritonitis; the duodenal ulcer and ulcers of the large intestine, traumatic lacerations of the stomach and intestines; also the important strangulations of the intestine, particularly herniæ with their sequelæ of permeability, infection, and necrosis of the paralyzed and inflated intestinal coils; ulcerations and pericystitic abscesses of the urinary bladder, purulent perinephritis, pus tubes, abscesses of the spleen, of the liver, of the pancreas, of the lymph-glands of the abdominal cavity; the spleen and glands may also come into question, particularly after enteric fever, the former after severe malaria. In the newborn the navel or the umbilical vein is sometimes the point of origin for septic infection; in young children ulcerative prolapse of the rectum. Finally, we may mention inflammations from the pleura and pericardium by way of the diaphragm; here, particularly in infancy, the pneumococcus frequently plays a rôle.[1]

Peritonitis originating from the vessels has already been mentioned,—that starting from the umbilical vein of the newborn; in a similar manner septic thromboses from branches of the vena cava and from the portal vein, usually the direct result of neighboring suppurations, may arise and cause disease of the peritoneum, more rarely septic emboli from the mesenteric arteries in general sepsis. Infection of the peritoneum by circulating toxins from the blood without local vascular disease has a special action,—certainly it is extremely rare. That which was formerly confounded with

[1] *O. Vierordt*, "Die Natur und Behandlung der Pneumokokkenempyeme." *Deutsch. Arch. f. klin. Med.*, Bd. LXIV.

it must be due to some neighboring focus in the peritoneum that may not even be detected in a careful autopsy; thus "typhoid peritonitis" is explained by the finest intestinal perforation, or the permeation of the serosa of the intestine without perforation, or by an abscess in the lymph-gland, etc.; malarial peritonitis by a focus in the spleen, and the septic form by a small vascular thrombus, etc. Only in acute articular rheumatism does it appear certain that the toxin passes from the blood into the peritoneal cavity as well as into the joints and all the serous cavities.

Chronic affections of the abdominal organs, such as tuberculosis, actinomycosis, gonorrhea, will be discussed later.

A review of this array of dangers which threaten the peritoneum reveals to us the extraordinarily varied nature of the actual originators of inflammation: The chemically high-graded and toxic gastric contents, the intestinal contents with their chemical substances and putrid bacterial admixture, from which, if sufficient time be allowed, pathogenic microörganisms are first cultivated in the inflammatory fluid; the appendix, of which almost the same might be said, but which also in its wall or in its lumen furnishes more or less pure cultures of pus organisms; the many other suppurations in which the attack sometimes appears to result from a gradual change of the bacteria to the highest degrees of virulence. Among the pyogenic organisms: Bacteria coli with their very varying toxicity, streptococci and staphylococci, less frequently pneumococci, very rarely gonococci and typhoid bacilli; furthermore, however, bacteria of decomposition of all kinds, in part the anaërobic.

Various as are the products capable of producing inflammation of the peritoneum, and innumerable as are the roads by which they may reach this tissue, everywhere, however, two circumstances especially influence the severity of the inflammation: The activity of the germ of inflammation and the rapidity with which it reaches the peritoneum. In regard to the latter, except in external trauma, it is nowhere greater than when one of the numerous hollow abdominal organs perforates into an entirely unprepared, that is, normal, peritoneum. Extreme examples of this kind are traumatic lacerations of the stomach and intestines, ulcer of the stomach, the duodenum, or small intestine, soon rupturing into the serosa and extending far down into the depths of the abdomen, a completely gangrenous appendix inflamed by the pressure of a fecal calculus and necrotic, in which rapid thrombosis of the vessels has occurred, or an empyema of the appendix or gall-bladder rupturing from the pressure of the eroding pus. Here usually large amounts of the contents of these organs are suddenly discharged into the free abdominal cavity; they unfold local and general toxic effects, and produce such similar clinical pictures that their differential diagnosis may occasion the greatest perplexity. And yet we are dealing with very different factors which have entered the peritoneum:

in perforation of the stomach, with the chemical constituents of the gastric contents and an admixture of bacteria in which specific pathogenic bacilli have from the onset certainly not been in the majority, and which—since we are almost always dealing with a preceding ulcer of the stomach—have usually lost much of their vitality and virulence under the decided influence of hydrochloric acid; in a rupture of the appendix occurring under the circumstances just mentioned, we are frequently dealing with a "putrid" infection in which besides the pathogenic bacteria the saprophytic also at first play an important rôle, and with substances which produce chemical and mechanical irritation. In other cases, naturally, within the lumen of the appendix or in its wall the bacterium coli or the streptococcus has developed to a high degree of virulence, and a more or less pure bacterium coli or streptococcus peritonitis develops. If, on the other hand, an inflamed purulent gall-bladder ruptures, a pure culture of less virulent coli bacilli enters the peritoneal sac with the pus, but nevertheless this also results in peritonitis.

Since the well known experimental investigations of Wegner, Grawitz and others, we understand that the animal flora of the peritoneum renders inert large numbers of microörganisms, partly by the bactericidal effect of its secretion, partly by the very rapid and intense absorption into the lymph-spaces, especially in the diaphragm. We know also, chiefly from the investigations of Grawitz, that chemical or physical substances which injure the epithelium diminish the protective power of the serosa, and it is this circumstance particularly which permits the development of bacterial peritonitis. These views have been based upon experiments and pathologic conditions both in animals and man. Clinical experience, however, if it be not too strongly influenced by the suggestive effect of these animal experiments, cannot escape the conviction that in human pathology the protective power of the normal peritoneum is not great. Certainly the surgeon, after the necessary toilet of a fibrino-purulent intestinal coil, sees the peritonitis disappear. But here we are not exactly dealing with a normal peritoneum producing and absorbing serum; but other curative factors are operative: Adhesions and the action of drainage. On the other hand, wherever in human pathology bacterial products of inflammation attack the normal peritoneal surface, there peritonitis generally develops very rapidly.

Valuable experiments by Silberschmidt[1] have shown that the combination of deleterious agents present in non-sterilized feces may, in from twelve to eighteen hours, cause the death of the experimental animal by purulent peritonitis. Only pathogenic bacteria are capable of producing a rapidly fatal diffuse peritonitis in rabbits, provided they do not find their way by a single injection but, being enclosed in an animal membrane

[1] *Silberschmidt, Mittheilungen aus den Schweizer med. Instituten.* 1. Reihe, Heft 5.

(fish bladder) *gradually penetrate to the free abdominal cavity.* Wieland [1] has conducted experiments of this kind; they are of great value in the study of these conditions; for they show that a minute change in the previous experimental arrangement is sufficient seriously to implicate the peritoneum which was at first so resistant, and to destroy the life of the animal; and, above all, they have the merit, as in Silbersehmidt's experiments, of closely resembling the conditions in human pathology; for, if highly virulent bacteria gradually approach and penetrate the lymph-spaces of the uterus or the wall of the appendix and enter the peritoneum, we actually have conditions similar to those in Wieland's tests.

In this connection let us glance over the history of Case No. 3, that of puerperal streptococcus peritonitis! It is a typical example of a diffuse fatal peritonitis due to a pure bacterial culture without the aid of "irritating substances"; and if, in accordance with the experiments of Grawitz and Wegner, we accept the theory of the attenuated resistance of the puerperal organism, we cannot readily understand it. Neither is this assumption borne out by the facts when a phlegmonous, erysipelatous inflammation of the wall of the appendix without perforation produces an extensive purulent peritonitis in the normal peritoneum (notwithstanding its undoubted powers of resistance).

The result of all this is that we must not estimate too high the power of the normal peritoneum to resist microbes; i. e., above all, to destroy them by absorption; if this be regrettable, it also has something in its favor. For we know and can prove by the previous histories that a massive absorption of toxins from the peritoneum leads to the severest form of peritoneal sepsis, and this we shall later discuss.

Protection by the normal absorption of the peritoneum, therefore, does not play the important rôle in pathology which was formerly often ascribed to it. *All the more important are the protective functions of the inflamed serosa.*

Before the peritoneum becomes implicated by inflammatory processes in its vicinity, changes are produced which may best be compared with the changes in the skin and subcutaneous cellular tissue surrounding a pus focus. In the peritoneum hyperemia develops, also a fluid exudate generally deficient in cells, which here, however, flows into the free abdominal cavity; this exudate may be entirely devoid of bacteria, in which case to-day we speak of a "chemical peritonitis"; rarely in these cases does the exudate become rich in cells, or even purulent. There is no doubt that these fluid effusions are of some value in rendering inert the phlogogenic microbes which enter the abdominal cavity from the focus of inflammation, and eventually perforate.

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[1] *E. Wieland,* "Experimentelle Untersuchungen über die Entstehung der Peritonitis." *Mitth. a. d. Schweizer med. Instituten,* Bd. II.

But this action, as already stated, must not be estimated too high; all the greater, however, is the protective effect of plastic exudates. Their production is the especial property of the serous membranes; they may develop even a few hours after the beginning of an inflammatory irritation, and may appear as swelling and redness of the skin surrounding a pus focus already formed around an inflammatory focus which threatens to attack the serosa. These plastic exudates, however, confer protection by leading to adhesions, and later, by the extension of these, to adhesions of the serosa with neighboring serous surfaces. Preferably they unite the anterior surface of the stomach with the abdominal wall, the greater curvature with the colon, the gall-bladder with the colon, etc.; they produce membranes which usually extend from the intestine at a point opposite the mesenteric attachment to the abdominal wall; they form a sac around the inflamed appendix which, if ensuing perforation occur, is calculated to absorb the putrid masses and the pus which these develop.

But, rapidly as these structures form, they nevertheless require time to attain the necessary degree of firmness.

Concerning this point, the clinical histories above cited should be investigated, the autopsy findings in the rapidly fatal cases, Nos. 2 and 3, and the findings at the operation and at the autopsy in cases Nos. 1, 4, and 5. The great importance of this circumstance is obvious, and since adhesions which exist before the rupture, if firm, afterward become stronger, sometimes in a very short time, we find perfectly firm encapsulated pus cavities in various regions of the abdomen; these encapsulated pus foci are probably most conspicuous alongside the cecum, behind the stomach as subphrenic abscesses, and in the true pelvis.

It is quite possible that occasionally even in perforation,—for example, of an ulcer of the appendix with a normal peritoneum, therefore, in the free abdominal cavity,—these adhesions may form rapidly enough to encapsulate putrid masses; it must here be taken for granted that these are quiescent, and this above all presupposes absolute quiet on the part of the patient and absence of motion in the intestine. Probably, however, these adhesions are generally preformed before the perforation; naturally, these are facts beyond our knowledge.

The more infectious the pus that has formed, and the greater the pressure under which it develops, the tougher must be the adhesions to protect the free abdominal cavity.

Let us again review cases Nos. 1, 2, and 5; we see that at first typical encapsulation took place; later, however, the adhesions were not compact enough. They either ruptured mechanically by pressure of the pus, by peristalsis or other causes, or were obliterated by the functions of the white cells and bacteria, and thus, finally, they paved the way for the entrance of inflammatory products into the free abdominal cavity, provided that, in the meantime, additional adhesions had not formed. In the

former case a general peritonitis secondarily developed, in the latter case there was a relapse of circumscribed peritonitis. In both cases we see the condition which Mikulicz has called the *progressive purulent form* of acute peritonitis.

The occurrence of such secondary ruptures depends, therefore, partly upon the relation between the internal pressure of the abscess and the firmness of its wall, and partly upon vital and chemico-physical processes in the wall itself.

As auxiliary factors of decided importance the intensity of intestinal and gastric peristalsis, possible external shock, etc., injury by purgatives and enemata or even by inunction or massage, must be considered. At all events, it is clear that the firmness of the boundaries is always doubtful, and, especially at the onset of the disease, this can hardly be looked upon as certain, least so with a closed abdomen, and this is of significance in the prognosis of such abdominal abscesses. The clinical history of Case No. 5 is an illustration of the surprises that may be in store for the physician.

This point will be further elucidated under the discussion of the treatment.

Chemical peritonitis has been referred to; this exhibits all the peculiarities of sero-fibrinous peritonitis, it may become rich in cells and even purulent—but bacteria are absent. It is probably brought about by bacterial toxins conveyed from a near focus; chemical peritonitis, therefore, occurs in the surroundings of an insignificant bacterial pus focus of the abdominal cavity; that is, from a periappendicitis, a psoas abscess, an inflamed appendix, an ulcer of the stomach or even an inflamed gall-bladder. In the latter case, as we have seen, it is generally plastic. Its appearance in the hernial sac about the incarcerated intestinal coil is characteristic and instructive; here, at all events, the paralyzed intestine is permeable for the toxins of intestinal bacteria and other soluble products, and this is the precursor of permeability for bacteria, and leads to necrosis of the intestinal wall; similar conditions arise in severe traumatic contusion of the intestine.

CHEMICAL PERITONITIS

In *chemical peritonitis* one thing is especially interesting to the clinician: The hyperemia and resulting fluid exudation appear and disappear *according to the focus of origin.* A circumscribed periappendicular abscess may produce a distributed chemical peritonitis in its surroundings; this manifests itself by moderate or even decided sensitiveness upon pressure and distention of the abdomen, and, perhaps, by decreased but, clinically, often threatening symptoms of extensive peritonitis; if the abscess is opened or ruptures spontaneously, these symptoms suddenly disappear. Much of that which the clinician, for want of a better term,

has designated as "peritoneal irritation" belongs to the realm of chemical peritonitis. This dependence of chemical peritonitis upon a bacterial focus of origin is due to the fact that the process, being independent of bacteria, does not generate new causes of inflammation, *is not in itself capable of propagation, or even progressive.* This does not preclude its occurrence as an accompanying process of great intensity and extension and directly or indirectly extremely dangerous (by moderate meteorism, forcing upward of the diaphragm, etc.). This form of inflammation differs from the metastases containing bacteria of peritoneal inflammation, which may spontaneously disappear provided their action is moderate and they may thus be destroyed by the peritoneum; on the other hand, in many cases they may survive and continue to develop even after the focus of origin has been removed. These points give us important indications for the treatment.

It is true the plastic exudates of chemical peritonitis, as well as those generated by the presence of bacteria, may persist for a long time; they may be organized into pseudo-membranes and indurations long after the source of inflammation has been removed. This newly formed connective tissue, by its bulk, resistance, and indistensibility, even by its cicatricial retraction, may contract, deform, and limit motion, and in various ways may narrow and constrict the abdominal organs and cause adhesions. In the consideration of chronic peritonitis we shall revert to this subject again.

The foregoing description makes clear the meaning of our statement that the severity of peritonitis depends primarily upon two things: The activity of the septic microörganisms and the rapidity with which they reach the serosa from a focus near the surface of the peritoneum.

In contrast to these numerous methods of infection from neighboring organs are invasions of the peritoneum by means of the blood or lymph-channels, which are certainly rare. They occur in acute articular rheumatism and occasionally in sepsis. In the cases in which small encapsulated empyemata, particularly the metapneumonic, complicate a purulent peritonitis, transmission by means of the blood may also sometimes occur, as I have previously stated.[1]

PATHOLOGY AND GENERAL CLINICAL PICTURE

In regard to the pathology and general clinical picture of acute diffuse peritonitis, the clinical observations quoted at the beginning of this article indicate distinctly that two different pathologic conditions are to be considered: A congestive inflammatory general condition with specific accompanying symptoms of peritoneal irritation, and the general picture of collapse. Mixed forms between these occur, although rarely; as a rule,

[1] *O. Vierordt, Deutsch. Arch. f. klin. Med.*, Bd. LXIV.

they may be sharply differentiated, but occasionally they rapidly merge into one another.

The triad of symptoms mentioned in the first clinical history,—severe abdominal pain, fever and vomiting,—has been sufficiently described in the epicrisis of the clinical histories, as well as peritoneal meteorism, which almost always occurs as a fourth symptom. These four symptoms, while not invariable, are found so uniformly in most cases of acute diffuse peritonitis, and they dominate the clinical picture to such an extent, that without a history the status of the patient frequently gives no clue for the differential diagnosis of the origin of the affection. If the history does not aid us, this question under some circumstances cannot be decided; but, as a rule, there are differential diagnostic guides to which we shall later refer.

Regarding the second condition, the seeming collapse, in our clinical histories we note its appearance under very different circumstances: In Case 1 it developed twice from the previously existing congestive inflammation; the first time it disappeared after the salutary rupture of the periappendicular pus into the intestine, and the second time it was the cause of death; in Case 2 (perforation of the stomach) it appeared a few hours after the onset of the affection; here, post mortem, the massive contents of the stomach were found in the abdominal cavity, and the peritoneum was quite smooth and glistening. In puerperal septic peritonitis collapse occurs early, but appears to be modified by the delirium and marked somnolence; post mortem we find a severe, purulent streptococcus peritonitis, but no perforation, no putrid infection.

The symptoms common to these clinical pictures are severe cardiac and vascular weakness, prostration, cold sweat, lowered general and surface temperature, and comparatively mild subjective symptoms; in addition to these a severe, widely distributed peritoneal irritation is common, and also probably the absorption of toxic products.

But, upon closer investigation, these conditions differ from each other, and far more etiologically than clinically. In one case we have the sudden flooding of the peritoneum with the gastric contents: Gastric juice containing hydrochloric acid, albuminates, peptones, salts, etc., and also bacteria which are of diminished vitality, consequently bacterial toxins here play no rôle. In the two other cases we have severe bacterial peritonitis—one being a pure streptococcus peritonitis; in both, especially in the latter, there is profuse absorption of bacterial toxins: Sepsis.

At the present time these conditions, especially in Germany, are regarded from too one-sided a standpoint,—usually as peritoneal sepsis,—and their other aspects are ignored; namely, the effects of shock upon the central organs, that is, the cardiac and vascular centers, originating from decided irritation of the sensory end organs of the sympathetic in the peritoneum, not alone in peracute perforations, but also in peritonitis

which rapidly becomes diffuse; and secondly, the internal congestion of the enormously dilated arteries of the splanchnic region which certainly play an important part; anemia of the rest of the body, giving rise to cardiac and vascular weakness, and probably also to dryness of the tongue and the marked thirst of which these patients complain, is not only due to the peritoneal irritation but is produced by the intoxication itself.

In the treatment it is advisable, and sometimes necessary, to observe the varying genesis and complicated nature of these clinical pictures at the bedside. Certainly intoxication, most often the septic form, plays an important part; the picture produced by the latter may also be of high substantive value in the diagnosis; for example, in acute intestinal occlusion, it points to strangulation, the immigration of bacteria through the paralyzed intestinal wall, and beginning septic peritonitis. But prolonged shock and congestion in the splanchnic area produce very similar symptoms, and neither the state of the sensorium nor the preceding fever is a positive and unmistakable guide to the differentiation. This can most readily be made from the manner of development of the affection and from the accompanying circumstances, and particularly from the diagnosis of the origin of the peritonitis.

The local symptoms of acute diffuse peritonitis have been sufficiently described in the epicrises of the clinical histories. But a few points should be emphasized.

LOCAL SYMPTOMS

Meteorism rarely develops at once; in some cases, particularly those in which gastric perforation or severe traumatic intestinal rupture occurs, also sometimes in duodenal perforation, it may appear very late or not at all. The abdomen is at first as hard as a board, tense and even retracted; often there is tearing pain. In the main, however, meteorism is typically uniform and rigid, individual inflated coils are nowhere seen, the dull tympanitic note is occasionally uniform, but nevertheless shows slight differences resembling those of the lungs (with marked tension!); we note the high position of the diaphragm, and the marginal border of the liver. We must bear in mind the necessity of frequent investigations for dulness so far as the state of the patient may permit; auscultation often reveals absolutely nothing,—"death-like silence in the abdominal cavity." Not rarely, however, soft, highly tympanitic, intestinal splashing is here and there heard, especially if slight diaphragmatic respiration is present, also soft friction sounds over the liver, the spleen and the upper portions of the intestine.

In gastric and intestinal perforation, *air in the peritoneal sac* is sometimes very easy to diagnosticate, particularly if it rapidly forms in large amounts, distends the belly-wall and raises the diaphragm, causes the liver dulness completely to disappear, and produces a clear, absolutely uniform,

more or less tympanitic note upon auscultatory percussion, with the absence of all signs upon auscultation, or profuse splashing sounds. Whether, owing to its importance in the diagnosis, we should try by cautious movement of the patient to obtain a succussion sound, can only be decided by the individual circumstances and by the physician's own conscience. If marked effusion is present, this becomes still more obvious by its prompt motion upon change of position. If peritoneal meteorism develops slowly after a preceding, high-graded, intestinal meteorism, it is difficult to discriminate between these signs; a uniform percussion note, the disappearance of liver dulness (even *complete*), the death-like silence which accompanies the latter, the soft, faint splashing, and even the unmistakable signs upon auscultatory percussion are manifest at quite a distance. The physical signs in both cases are very similar. The differentiation may be just as great when the gas in the peritoneum owes its origin not to perforation but to the putrid decomposition of a fluid exudate.

Peritoneal meteorism which appears rapidly proves perforation; inversely, however, it may be absent in perforation; it is most frequent in the perforation of typhoid intestinal ulcers, and relatively rare, at least at the onset, in complete traumatic intestinal rupture; perforation of the appendix seldom causes the discharge of gas; in gastric perforation this varies greatly.

As has already been stated, diffuse peritonitis is by no means always distributed evenly and "diffusely"; the large omentum and the transverse colon frequently act as a barrier; appendicular peritonitis, peritonitis of the small intestine, and puerperal peritonitis chiefly originate beneath this barrier, gastric peritonitis develops above it, and it is therefore important for us to decide as to the starting-point of a peritonitis, whether it presents itself as diffused inferiorly or diffused superiorly. The clinico-diagnostic difficulties of this differentiation will be found great beyond expectation. The symptoms of subdiaphragmatic peritonitis, absence of diaphragm respiration, and singultus are not strictly confined to this form. I remember quite a number of autopsies in cases which had shown these symptoms during life, and the entire upper portions of the peritoneum were free. The persistence of diaphragmatic respiration might be considered to indicate a superior peritonitis, but diaphragmatic respiration is, in fact, almost always absent from the onset in the diffuse forms.

The circumstance is very important that in gastric, and partly also in duodenal, perforation, vomiting is insignificant or may be entirely absent; frequently there are eructations. In contrast to this, in deep-seated peritonitis urinary difficulties and a high degree of pain upon pressure are observed during "cautious" vaginal and rectal examinations.

With gas in the abdominal cavity peritonitis in the upper part is mostly diffuse; gas everywhere finds its way upward, and promotes the rapid distribution of inflammation.

DIAGNOSIS

The diagnosis of diffuse peritonitis may be easily made when we consider the totality of the symptoms. In a minority of cases, however, we meet with differentio-diagnostic difficulties which are usually very great.

An acute peritonitis without meteorism, i. e., with a hard retracted abdomen, with excruciating pain in the gastric region, but without vomiting or constipation, and in which there is perhaps a history of ulcer, may simulate a severe cardialgic attack in a patient with gastric ulcer; fever, diffused pain upon pressure in the hypochondriac region, and even in the lumbar region, and the symptoms of collapse will favor peritonitis, but the diagnosis may remain doubtful until meteorism appears. The same conditions are also observed in ulcer of the duodenum.

Severe inflammatory *attacks of gall-stone colic* with a distended abdomen, vomiting, and even fecal vomiting and fever, may cause great perplexity in the differential diagnosis, particularly if the anxious patient states that the whole abdomen is painful upon pressure. The difficulty is, however, at least lessened by the rapid disappearance of meteorism when the attack ceases, and perhaps with the administration of morphin.

In the majority of cases, however, it is difficult to decide whether peritonitis or *intestinal obstruction* is present, particularly if strangulation be suspected, since the latter, as is well known, gradually produces peritonitis. Simple obstruction is readily differentiated from peritonitis by the absence of fever, by the less painful meteorism, by a more or less distinct local peristalsis, by collapse which sets in late or does not appear at all. The obstacles are even greater in the diagnosis of obstruction due to strangulation: Examination of the hernial rings (they must all be examined, and all symptoms of hernia considered) by rectum will sometimes at once decide the question in favor of the latter; but where all these tests are negative or uncertain (as in long-standing, non-incarcerated hernia), in the absence of fixed inflated coils (v. Wahl) or of peristalsis above these (Sehlange)—as is unfortunately *frequently* the case—the differential diagnosis at once becomes very obscure; early fever favors peritonitis (initial shock occurs in this condition as well as in strangulation) and hemorrhagic dejecta indicate strangulation, particularly invagination. The longer the condition lasts the more difficult is the diagnostic differentiation, because peritonitis is then added to strangulation. It is obvious that under these circumstances the previous history of the patient is important and occasionally decisive.

The same conditions are met with in the extremely rare cases where an enormous fecal accumulation causes great meteorism, vomiting, sensitiveness of the abdomen, eventually fever, and simulates peritonitis. Uremia also may lead to confusion when accompanied by severe vomiting,

meteorism, diarrhea and fever, and when the symptoms of nephritis (acute or chronic) have been insufficiently observed.

PSEUDO-PERITONITIS, PERITONISM

By a pseudo-peritonitis, peritonism (Gubler), we mean conditions in which the combination of abdominal pain, vomiting, a tense and usually distended abdomen, constipation and perhaps even fever depending upon a hysterical basis simulates peritonitis. These cases usually occur in young hysterical persons, not rarely in children; occasionally the causes are mild intestinal and genital affections, retention of urine and feces, slight trauma. The clinical picture may, at the first glance, lead to confusion; but upon careful observation a certain disproportion in the symptoms becomes obvious; for example, tearing pain and continuous vomiting without a sign of collapse; in one of my cases there was severe spontaneous pain as well as tenderness upon pressure with marked meteorism, but, instead of lying still, upon palpation the patient made energetic efforts to rise; this does not occur in acute peritonitis. Other characteristics are the abrupt change in the symptoms, the effect of suggestion particularly on the pain, the absence of cardiac asthenia (although Naunyn saw dyspnea and a poor pulse due to the elevated position of the diaphragm), finally, stigmata and *antecedentia hysterica* must be considered, both of which, naturally, may be absent in the young. Taken all in all, the careful observer is liable to be mistaken the first day of the disease, but, upon the second, one of these signs will lead him to a correct conclusion.

In all of these cases, in which, in contrast to diffuse peritonitis, non-inflammatory or, at least, not diffusely inflammatory affections in the abdomen, or other conditions are to be considered, a rise in the rectal temperature (two or more degrees higher than in the axilla) may be of diagnostic importance, but only when the temperature is very accurately taken by the axilla.

The *diagnosis of the point of origin* is easy, provided the symptoms of the original affection have been previously observed by the physician; otherwise, the diagnosis is generally the more obscure the greater the development of the peritonitis. But even then it may often be made from the history—which, however, may be erroneous (cardialgia and gall-stone colic, ulcer of the stomach and ulcer of the duodenum, an affection of the pancreas, perityphlitis and genital affections, etc.).

It is most important to determine the seat of greatest tenderness, and there eventually to locate an objective point. Some authors have recently attached great significance to the hyperalgic zones of the skin. Examination of the hernial rings and of the vagina should never be omitted; both this and rectal examination are generally necessary for the detection of exudates into the pelvis, and to differentiate perityphlitis from genital and bladder affections.

It must be remembered that the individual abdominal organs, particularly the appendix, have no fixed position; the tip of a long appendix may reach far down into the right iliac fossa, as has been previously remarked. A perieholecystitis will occasionally be situated very near McBurney's point, or may be also somewhat displaced laterally. There is naturally no positive seat for perforation of the small intestine, although typhoid perforation is often found low down upon the left side. Ovarian affections may be widely distributed, particularly toward the ileo-cecal region. The pancreas and duodenum have fixed locations, but here the differentiation from the stomach is difficult.

CIRCUMSCRIBED ACUTE PERITONITIS WITH PARTICULAR REFERENCE TO PERITYPHLITIS

PATHOLOGY

Circumscribed peritonitis is prone to develop when bacteria find ingress to the abdominal cavity and penetrate to regions preformed by adhesions. We have previously seen that in perforation the severity of the infection of the abdominal cavity depends upon the amount of material discharged into it from the stomach and intestines, upon the entrance of pyogenic organisms from the lymph-tracts, upon the virulence of the pathogenic pure cultures which have been previously produced in the tissues. Now, if the perforation be very minute or the virulence but moderate, if it take place in an area favorable for deposit (the ileo-cecal region, the pelvis, the subphrenic space), on the cessation, spontaneous or induced, of intestinal peristalsis and when the patient remains very quiet—it certainly happens that a previously normal peritoneum without adhesions will produce direct epithelial or fibrinous adhesions of its walls, and, in the meantime, will absorb a certain amount of the poison and in this manner localize the process. The individual parts of the abdominal cavity joined to the mesentery form a fan-like structure which aids us here. The clinical picture depends upon how rapidly encapsulation takes place, whether it be complete or partial, whether the toxins do not produce (although their agents themselves do not) a diffuse chemical peritonitis beyond the protective wall, and upon the density of the adhesions, the firmness of their hold, and their further course. Naturally it also depends upon the reaction of the entire organism to the irritation of the peritoneum and to the intoxication.

From the different forms of circumscribed peritonitis I shall single out perityphlitis, and describe this affection in detail. This is the disease which, in the majority of cases, presents itself to the family physician, and it has become a mooted question whether it does not, from the very

onset, belong to the realm of surgery. Besides, it shows the greatest variations, and therefore best illustrates all of the conditions which play a part in circumscribed peritonitis.

We have seen from the history of these cases that circumscribed perityphlitis may be absent clinically and anatomically (Case 3) in the severest, i. e., in the most acute cases of perforation of the appendix; that it may sooner or later develop in diffuse perforation of the appendix (Case 1), and may even appear simultaneously with this or may precede it; that an accompanying general peritoneal irritation may dominate the clinical picture; that, finally, and fortunately in the majority of cases, it may occur alone and exclusively in an otherwise normal belly. In its *course* this focal affection may extend to the surrounding portions of the abdominal cavity. It has just been mentioned that perityphlitis may appear gradually as the actual nucleus of a distributed peritonitis, and when the peritonitis disappears may dominate the picture; inversely, from the originally circumscribed affection one or more serious relapses may unexpectedly occur, perhaps with fatal result.

Which of these extraordinarily different clinical pictures will develop depends chiefly upon the nature of the appendicitis which has produced the peritoneal inflammation (skolikoiditis is the designation proposed by Nothnagel); this collective name, however, includes many varieties. If we leave out of consideration the tubercular, the typhoid, and the actinomycotic affections of the appendix, most of the non-specific cases may be referred to two large groups of causal changes in the appendix: To *fecal impaction,* and to certain *chronic inflammatory* and also *acute "relapses"* of the wall of the appendix.

Proceeding from the most dangerous to the mildest diseases of the appendix, we first distinguish an acute distributed gangrene of the organ with immediate perforation and putrid flooding of the surrounding area; next, the milder cases, i. e., those with gradual onset and, at least in the beginning, less extensive putrid infection of the peritoneum by the perforating, gangrenous or non-gangrenous ulcer. These lesions are almost always due to the greatly dreaded fecal concrement, although it may certainly be absent. We recognize also a gangrenous inflammation of the appendix without fecal calculus, particularly in stenosis, followed by the rupture (which may be gradual) of an empyema of an appendix which is closed at the cecal end by stricture, but also by empyemata and suppurations of the wall of an appendix which, without a gross rupture, permits the passage of pus cocci; nearly all of these cases are due to collections of bacteria (Tavel and Lanz), among which pathogenic microbes that have previously attained decided virulence are found to predominate (almost invariably bacterium coli). But simple phlegmonous or erysipelatous inflammations of the mucosa of the appendix may, without themselves being purulent, produce a suppurative perityphlitis usually charac-

terized, at least at first, by a benign form, i. e., with slight toxic action and a tendency to limitation.

Passing from the severer to the milder forms, we note that the appendix is frequently the seat of "chronic catarrh" of the mucous membrane and its numerous glands and follicles (angina of the vermiform appendix, Sahli), in itself not serious, but also of deeper chronic inflammation of its connective tissue (appendicitis granulosa, Riedel). These changes may be the starting point of the above mentioned severe forms of inflammation, either directly or by causing secondary changes: Stenosis and stricture by loss of epithelium, the formation of cicatrix, adhesion of the surfaces of mucous membranes lying opposite each other.

A factor of great significance in the development of these forms of appendicitis is that the serosa of the appendix, and also its mesenteriolum, often early implicate the wall of the organ, even by mild chronic inflammatory changes. In these instances the serosa, as we learn from early surgical operations, is rarely quite normal, but is usually reddened, permeated by numerous vessels, more or less thickened, often unevenly, by the formation of constrictions of the appendix; frequently the mesenteriolum is also implicated, being shortened, thickened, bent and torn, causing immovability of the contractile organ. Often the entire mass early becomes embedded in new connective tissue, or the appendix at its end or at some other part of its length adheres to the cecum, the iliac fossa, the ovary or the bladder.

There can be no doubt that these inflammations of the mucous membrane and the early changes in the serosa indicate severe disease. If we remember that the appendix is comparatively long and thin, a hollow, secreting organ, it is not difficult to realize that even a moderate diminution of the elasticity of its enclosing serosa, a slight induration of the same, but still more an external stenosis and kinking, may very readily check the flow of its secretion, as well as lead to circulatory disturbances in its walls. Moderate stricture may act like a narrowed foramen upon catarrh of the frontal bone; desquamation and serous transudation of its tissue may also cause venous stasis; the finding of a penis-like thickening of the process, but even more the microscopic findings of Riedel (round-cell accumulation, disappearance of the glands and *hemorrhages*) must increase this presumption. But these processes take place in the neighborhood of products which mechanically and toxically injure them, and in the presence of putrid collections of bacteria; doubtless in the latter case the pathogenic bacteria which are always present frequently gain the upper hand. We must, therefore, on the one hand, attribute to the condition of the secretion, and on the other hand, to stasis combined with the above circumstances, a part in the genesis of all mild and severe forms of periappendicitis which develop without fecal calculi; but also the severest forms, for the remarkable finding of acute gangrene without fecal

calculus may be plausibly explained by a free desquamation of the organ with limited distensibility, by immediate severe stasis in the region of the fine venous branches of the interior, and by gangrene from the influence of bacteria constantly ready to develop.

The most vital point here is the intimate relation of comparatively mild changes in the appendix which cause no apprehension to the most severe and most dangerous. This is probably due to the fecal calculus which may remain a varying length of time in the appendix without producing symptoms (in some cases large fecal calculi have been discovered accidentally without resultant effect), and then, as a rule suddenly, exhibits decided activity. On the other hand, it may be due to the previously mentioned chronic inflammatory conditions. In regard to the latter we are indebted to Riedel for having systematically paved the way to the recognition of their relations to perityphlitis. He has shown that chronic appendicitis is in some cases a comparatively mild, in others an exceedingly grave, affection. He relates his observations and results of investigation so clearly that I cannot refrain from quoting his remarks on the point in question: "In one case the disease (appendicitis granulosa) leads insidiously and painlessly in the course of years to obliteration; in another, one or several inflammatory relapses occur and obliteration results; in a third the appendix, notwithstanding several relapses, remains in the state of appendicitis granulosa; in a fourth, the lymph-glands become infected; in a fifth, a periappendicular abscess forms which may later be absorbed without a trace of pus appearing in the appendix; finally, in a sixth, the acute inflammatory paroxysm begins with suppuration, even with gangrene; here appendicitis granulosa forms the basis for the acute suppurative attack, usually without fecal calculus, so that frequently the pus is but slightly or not at all putrid; with gangrene that sets in at once it is usually putrid, but not invariably."

If we consider what has long been known of fecal calculi, and what has become obvious from the conditions just described, we come to the conclusion that in by far the majority of cases of severe appendicular peritonitis dangerous to life the disease has existed for a longer or shorter time. For the clinician, however, the important questions at once arise: Can these preceding changes be clinically recognized, or can we, at least, approach more nearly to their recognition? These questions we will discuss later.

If suppuration occurs in any of the previously mentioned regions outside of the appendix, the development and course depend upon various circumstances. The most malignant forms are the putrid infections and those from highly virulent and more or less pure pyogenic agents (bacteria coli, streptococci and staphylococci), the former occurring in gangrene with or without fecal calculus, but usually without, the latter less frequently in a few cases of empyema and phlegmonous appendicitis.

An important and usually decisive factor is the eventual presence and the strength of the adhesions; coarse ones resist the mechanical and vital influence of pus, weaker ones succumb. As auxiliary conditions we must mention the position of the abscesses as related to the cecum: Those medially situated soon penetrate into the free abdominal cavity, those laterally situated toward the sacral hollow of the pelvis are most readily limited. Suppuration between the layers of the mesenteriolum appendicis frequently extends to the retrocecal space and occasionally along the colon as far as the descending colon; thus it may give rise to paranephritis either dextral or sinistral, subphrenic abscess, empyema upon the right or left side; and these may apparently be produced paroxysmally. Suppuration low down in the median line readily finds its way to the pelvis. It is obvious that when pus is found in this locality the original position of the appendix has a decided influence.

Periappendicular suppuration, even when not very extensive, produces desquamation and serous transudation of the surroundings: of the serosa of the cecum and appendix, the preformed adhesions and, possibly, the adherent parietal peritoneum in this region, perhaps also of some of the mesenteric glands; in some cases the connective tissue of the retrocecal space becomes involved; all this occurs to a varying extent, and produces the *perityphlitic tumor* in the interior of which suppuration is found which resembles the purulent nucleus of a furuncle (Sahli). If the pus becomes more profuse the tumor may show fluctuation; it may become enlarged by an accumulation of feces in the paralyzed cecum, by reflex action of the inflammation or by the intestinal paralysis which may be very extensive, and by vomiting. Occasionally it may produce distant effects: Engorgement and, not rarely, serous exudation ("chemical peritonitis") of the free abdominal cavity which is usually the result of a general toxic action, namely, fever.

As we have just seen, non-purulent inflammatory processes in the appendix cause external suppuration, mild irritative conditions in the vermiform process cause thickening of the serosa and adhesions. These mild irritations, especially the relapses of appendicitis granulosa, are probably capable of producing relapsing, that is paroxysmal, serous transudation and serous or sero-fibrinous exudates (Sonnenburg) in the surroundings without the formation of pus, and these are clinically observed as tumors; they are characterized by their brief duration, their rapid disappearance, sometimes by their sequelæ of marked thickening and adhesions.

The variety of conditions produced by circumscribed perityphlitic suppuration is partly illustrated by clinical histories; left to themselves, after more or less time they are prone to rupture, generally into the intestine, especially into the colon, and then heal; they may also rupture externally,—through the skin, into the bladder, into the rectum, into a pleural

cavity and perhaps into the lung; or they penetrate to a greater or less extent within the free abdominal cavity and immediately cause an acute diffuse peritonitis, or, in case adhesions were preformed, a progressive purulent peritonitis (compare clinical history No. 4); in the series of relapses they may produce here and there a number of new foci which result in a more or less rapid destruction of the organism. But metastatic suppurations may also, particularly by rupture through the intestine, force their way outward, and surprising recovery has been observed even in the severest cases; in the first stages of progressive peritonitis, however, we often note a sudden halt in the process, it ceases everywhere except in the perityphlitic center, and even here it finally runs a favorable course or becomes chronic.

Besides this unexpected change for the better there may be an unlooked-for change for the worse; a perityphlitic abscess which has previously been circumscribed may, at any time, after days or weeks, rupture; general peritonitis, sepsis, and death follow. Doubtless this occurrence is most frequent in recent encapsulation, i. e., in perityphlitic abscesses which have existed only three to five days. The longer this period and the firmer the encapsulation, the more unlikely is a fatal rupture; but it can never be excluded with *certainty* while pus is present in the abdominal cavity. It is in putrid suppuration that this condition is always most to be feared.

There can be no doubt that on partial discharge of the pus, for example, after surgical operation, the remaining pus is absorbed, even if the wound be imperfectly drained. Consequent upon operation special conditions are present; congestive hyperemia probably acts by vitalizing all important processes, the absorbent as well as the bactericidal. Therefore in the general application of these observations, particularly when we have the condition of a closed belly, we must be cautious; prior to operation, such a condition is extremely difficult to distinguish. What appears to be absorption may very likely be an unperceived rupture into the intestine. Even more than in the normal peritoneum, we must avoid over-estimating the absorptive activity of the peritoneal pseudo-membranes. We have little certain knowledge regarding these; all is conjecture.

Of other complications produced by pus, only septic thromboses of venous trunks in the portal vein system, or of branches of the iliac, or of this vessel itself, are to be mentioned. The latter may lead to septic embolism of the pulmonary artery, and pulmonary abscess (not always fatal). These thromboses usually result in general sepsis and death.

SYMPTOMS

In considering the clinical symptoms of circumscribed perityphlitis, we must again review the clinical histories: Acute illness with severe pain in the ileo-cecal region, vomiting, a varying temperature and usually

rapidly rising fever, but rarely fulminant symptoms; frequently, at first, a few thin fecal movements, followed by constipation which depends upon intestinal paralysis, and then the appearance of a painful, more or less immovable tumor in the region of McBurney's point, which at first increases in size, is coarse upon palpation, but rarely fluctuates markedly; the inguinal glands are occasionally swollen; flexing the right leg often produces deep-seated pain in the belly; if this be very severe it favors paratyphlitis. This is the typical picture to which, under some circumstances, the more or less intense symptoms of a diffuse peritoneal inflammation or "peritoneal irritation" are added.

The case is not always so simple; in all similarly located affections the possibility, and even the likelihood, of perityphlitis will come into mind because this disease is so frequent. But the symptoms will often necessitate careful inquiry; under some circumstances, nothing more can be found than an indistinctly limited resistance deep in the belly and covered by an intestine containing gas,—occasionally not even this.[1] To ensure certainty from the onset in the differential diagnosis an examination must always be made of the hernial rings, of the vagina, as well as of the rectum; vaginal as well as rectal examination is usually to be repeated in the further course (to solve the question of possible descent into the pelvis).

Pain, fever and resistance are the principal symptoms.

Pain.—The pain in the majority of cases at once correctly indicates the location; as a rule it is spontaneously severe and upon pressure unbearable; it is situated near McBurney's point or often somewhat below it. At the onset it may be diffuse, or may be complained of in a paradoxical region (upon the right above, upon the left below, etc.); but, among all the acute abdominal affections with pain, it should be remembered that perityphlitis is the most frequent; it should always be borne in mind. The position and the radiation of the pain from the median line downward, and often associated with dysuria (indicating inflammation toward the pelvis), to the lateral and posterior lumbar region (inflammation in the retrocecal and retrocolic space) are only briefly mentioned. Psoas pain upon flexure of the right side certainly occurs in perityphlitis without decided paratyphlitis. Painful flexure contraction of the thigh, however, and the impossibility of extending it, point with great positiveness to the retrocecal connective tissue. Lancinating pain upon pressure points to pus, dull pain does not exclude it. Very severe spontaneous pain and

[1] After this article was finished I read an excellent publication by B. v. Beck upon "Weitere Erfahrungen über operative Behandlung der diffusen eitrigen Peritonitis" (*Beiträge zur klinischen Chirurgie*, 1902), from which I see that v. Beck, in the diagnosis of the point of origin of peritonitis, attaches importance to the previously mentioned circumscribed hyperalgic zones of the skin; he looks upon them as a valuable indication. In regard to this symptom, I have as yet no experience.

pain upon pressure with only a small tumor and mild general symptoms are not rarely observed in empyema.

Fever.—In regard to the fever, I should like to state briefly that in my experience high fever, according to its duration and type, enables us to arrive at positive diagnostic and prognostic conclusions. But the absence of fever, or a fall in the temperature, must be utilized with a certain reserve. A glance at the clinical history shows that high fever, setting in abruptly with chills, with a parallel increase in the pulse rate, here, as well as frequently in pathology, indicates pus; yet it may occur without pus, particularly in the inflammatory relapses of appendicitis granulosa (Riedel); less rarely is vomiting absent in either case. High fever with a very rapid small pulse, excruciating pain, and severe vomiting usually points to perforation. It will be remembered, too, that the symptoms of septic collapse and the general abdominal signs of perforation occur conjointly with immediate diffuse peritonitis or peritoneal sepsis; when the exudate is limited, severe septic collapse is absent and the abdominal symptoms are slight, or, in case they are well developed, they decline in one to three days, usually with a fall in temperature. But cases midway between this latter picture and that of acute septic peritonitis are not rare. The fever course which indicates rapid limitation is, therefore, that which gradually declines for three, five or more days after the initial height. The local signs accompanying this are usually those of concentration upon the perityphlon. A change at this time with rise of fever and still greater distribution of the process is serious, but, fortunately, infrequent. Gradual limitation corresponds to a sub-continuous range with diffuse abdominal symptoms lasting several days, the fever disappearing by lysis when the limitation has been reached; but even complete circumscribed peritonitis sometimes gives rise to high fever continuing for a week or longer. If collapse is not present sudden decline of the fever almost always indicates perforation of the abscess into the intestine, or one of the other directions indicated. The feces and urine should be watched!

A milder type of fever usually accompanies slight perforations of the appendix or purulent periappendicitis without perforation, but also appendicitis simplex with slight, non-purulent implication of the serosa. Empyema of the appendix presents a varying temperature, usually not high fever, occasionally none at all; sometimes, however, chills and great elevation of temperature are observed.

In the later course a sudden, sharp rise without symptoms of sepsis points to an increase of the purulent inflammation; that is, in accordance with the conditions present and the accompanying local symptoms, to progressive purulent peritonitis, to an extension of the process along the colon, or to subphrenic abscess, empyema, etc.; another decided rise with immediate septic symptoms which at first may be only latent, indicates

threatening diffuse peritonitis. But even here there are also intermediate forms: For example, rapid and progressive distribution of the disease without septic collapse.

The long-continued, progressive purulent forms naturally exhibit a long-continued febrile state with great variation in temperature, chills, a sub-continuous course during renewed encapsulation, a decline with possible rupture, etc.

I cannot go beyond these general remarks; it is easy to construct medium courses as types; Rotter and others have no doubt constructed these by carefully utilizing their numerous observations. From a didactic standpoint these types, which are exemplified only in a certain number of cases, are always questionable, because the reader who does not bear in mind the multiplicity of exceptions is always inclined to adhere closer to the type than does the author himself.

Tumor.—The tumor varies greatly in size, varies in position according to its mode of attachment to the appendix and its length, varies in consistency according to the manner of its production. Its walls may be perceptible or may be overlapped by intestinal coils, and in the latter case, if meteorism be present, may be completely obscured. Palpation as well as deep percussion [1] is restricted by the pain of the patient, as well as by prudence. Experience has often revealed to surgeons empyemata and abscesses which were at the point of rupture. Usually, however, it suffices for the diagnosis if we discover resistance over the cecum, or medially or laterally related to it. We may be fully assured of a firm limitation when a coarse but slightly painful tumor is found, and also when a large tumor, with a parallel retardation of the other symptoms, daily becomes smaller and more compact; yet these findings afford no guarantee against secondary perforation.

Vomiting.—Vomiting is due to reflex irritation of the peritoneum; in the malignant infections which are rapidly and widely distributed, it is often severe with the ejection of grass-green masses; in high-graded intestinal paralysis obstruction occurs under some circumstances, usually with the symptoms of collapse.

These forms are typical of acute diffuse peritonitis. In the circumscribed form the vomited material is rarely grass-green, but the vomiting is frequently severe and distressing, and often bears a certain relation to the intensity of the inflammation. Vomiting usually accompanies relapses of appendicitis, and especially when they run their course without purulent inflammation of the serosa; this symptom is met with, therefore, in many cases when all other signs of implication of the peritoneum are absent.

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[1] According to Weil, I understand by deep percussion that form in which the plexímeter or the plexímeter finger is pressed deeply into the belly.

Urine.—The urine varies, but these variations do not always correspond to the severity of the disease; in the severer cases, particularly those with high fever, it often contains albumin and indican; in children these, as well as aceto-acetic acid, are found much more frequently and often in decided quantities. Dysuric symptoms depend upon implication of the lower portion of the abdominal cavity; they may be temporarily observed when no other symptom points to the areas surrounding the bladder.

DIFFERENTIAL DIAGNOSIS

1. The Differentiation of Perityphlitis from Other Conditions.—In case we find a distinct tumor between the umbilicus and the anterior superior spine of the ilium the question of a fecal tumor comes into consideration. It must be remarked that this may be an accompanying symptom of perityphlitis, and, if accompanied by severe pain and vomiting, must always be borne in mind. A mere accumulation of feces also occurs here, but is much rarer in ordinary constipation than in local conditions which cause stasis, stenosis of the colon low down (tumor of the left flexure, cholecystitis with adhesion to the transverse colon), or stenosis at the cecum itself from old adhesions or thickening of the serosa, i. e., residues of a *former* perityphlitis; such a fecal tumor is moderately sensitive, movable, circular, usually compressible, and disappears after a bowel evacuation (as a matter of caution this had better be produced by enemata).

Neoplasms of the cecum or colon may, under some circumstances, also cause tumors in this region; these are also movable at first, are very coarse, non-sensitive or slightly painful. Here attacks resembling perityphlitis are very rare; paroxysmal marked stasis is produced much more readily; during the attack the diagnosis may occasionally be difficult on account of insufficient preceding observation.

The latter is also true of acute intestinal occlusion in this region, particularly that due to strangulation; invagination of the ileum into the cecum causes at first a characteristic tumor, later hemorrhagic dejecta; the condition cannot be readily mistaken.

Tuberculosis and actinomycosis of the cecum and appendix, particularly in the earlier stages, may lead to extreme differentio-diagnostic perplexity.

Besides appendicitis, other pathologic conditions produce painful attacks in the vicinity of McBurney's point, pain upon pressure, vomiting and fever, and these are: Cholelithiasis or choleeystitis, right-sided renal colic, particularly when combined with a dislocated kidney, inflammatory disease and tumors of the ovaries and of the tubes, gravitation abscess from the psoas. Here a careful history is of great value; not rarely the diagnosis can be made at once; a large liver and the respiratory movability of a tumor that may be present favors pericholeeystitis; we must be

eautious, however, in the consideration of moderate jaundiee, since it also oeeurs in perityphlitis; gall-stones are rarely found in the feces, but, if present, they aid in the decision. This differential diagnosis is sometimes difficult or even impossible. If the kidneys are implicated, it will be evident from the well known manifestations of a disloeated kidney as well as the examination of the urine (nephrolithiasis). The question of disease of the female genitalia will usually be decided by a vaginal examination which should never be omitted in any case; naturally this is not always decisive, and especially when there is implication of the serosa of the pelvis in perityphlitis, or in case of simultaneous disease of the adnexa and perityphlitis. When considering gravitation abscess, the vertebral column and the sub-inguinal region should be carefully examined; the history is of value in this instanee as well as in all others.

The most difficult cases are those in which no resistance is present and the pain ean scarcely be localized, or where pain and resistance are present in a paradoxical area, as in the case of a very long appendix adherent at its apex; here the focus may be under the liver, in the pelvis, even upon the left side of the abdomen, and the diagnosis of appendieitis may cause the greatest perplexity. In all such instanees, if the accompanying symptoms point to appendieitis, this affection should be first considered as it is relatively the most frequent. In late childhood it is of especial importance, as disease of the biliary passages and gastric ulcers seldom arise in these subjects; they occur at the end of the second decade of life, but do not often occasion symptoms that resemble appendicitis. From the thirtieth year upward the frequency of appendicitis diminishes (i. e., of the first attack!) and after the fortieth year the disease is rare. Hernia and internal incarceration must invariably be thought of: History and local examination including that per rectum!

2. **The diagnosis of the form of perityphlitis** is the most difficult part of this entire subject.

I must first emphasize that appendicitis in itself, particularly that form with but slight implication of the serosa and, at all events, without purulent implication, is very difficult to differentiate from periappendicitis. The affection begins with mild fever of brief duration, sometimes, however, with a decided rise in temperature and ileo-cecal pain; nausea and vomiting invariably point to implication, or at least to congestion, of the serosa; constipation may also be present. Local examination reveals pain upon pressure and in some of the cases a tumor, corresponding to the swollen appendix, globular in shape and often distinctly movable, sharply circumscribed, or apparently embedded in a firm mass—serous exudation in the serosa, in which case implication of the peritoneum may be assumed with certainty. This condition has long been recognized but was more accurately studied in the operations which Riedel performed early in his cases, and he explains it as an inflammatory, phlegmonous, or erysipela-

tous relapse of chronic appendicitis granulosa hemorrhagica. It is often the precursor of well developed attacks of perityphlitis. The diagnosis may be made with great probability when, besides the symptoms already enumerated, no sign of severe general implication, no special meteorism, no symptoms attributable to the lower portion of the abdominal cavity are present, and no large tumor has formed. It is well constantly to bear in mind that a perforative peritonitis may set in insidiously, and for this reason the patient must be carefully watched.

From what has been stated the difficulty of the differential diagnosis of the *forms of perityphlitis* may be imagined. Clear clinical diagnoses which include the anatomical condition of the appendix, the cecum, the peritoneum, etc., and besides convey an idea as to what may develop, can only be made in exceptional cases. For the present we must be content with a diagnosis which is made principally from the practical standpoint of prognosis and therapy; and here the burning question always is: Is the process localized, and will it remain so?

If no pus is present the attack as such may be looked upon as mild —with rare exceptions which shall at once be mentioned. I believe it likely that in the, fortunately, very rare cases in which a severe, diffuse, septic perforative peritonitis (with fecal stone always present) sets in with a stage of several days of mild fever, pain, and moderate vomiting— in these cases in the "prodromal stage"—nothing more is present than a simple appendicitis preceding gangrene. These are exceptional cases which we are unable to recognize by any method of our art. In general the law holds good: If there is no pus the case is mild, if pus is present it is serious, and when the pus is putrid, situated around or in the appendix, the condition is extremely serious, i. e., immediately dangerous to life; in the latter case perforation almost invariably takes place, and is usually due to fecal calculus; rarely does a putrid empyema form.

The question, therefore, arises: Can we diagnosticate the presence of pus, and can we recognize it as putrid?

Not long ago the answer to this first question appeared to be greatly simplified by the assumption that every perityphlitic exudate contained a purulent nucleus (Sahli); it *appeared* simplified—yet, from the practical standpoint of prognosis and from the treatment which was to be undertaken or rejected it nevertheless was not so—because the presence of pus is of very varying importance (compare page 544). But we are not always dealing with pus: We learn from the early operations, above all, those of Riedel, that there may be a serous, or sero-fibrinous, periappendicitis, and a recently inflamed appendicitis surrounding this.

Therefore, in an acute attack of appendicular disease, we are confronted by the question: Is pus present or not?

A fluctuating tumor appearing acutely in this region always indicates pus; a small tumor will by its resistance at least permit us to assume

its fluid contents, although exploratory puncture is very rarely permissible; if any fluid be present, it is pus. But in the majority of cases we find firm tumors or tumors with more or less indistinct resistance. In positive tumors with firm walls an exploratory puncture may be made; but it does not in all cases show pus; this is only the case when the point penetrates to the purulent nucleus. In all cases of resistance and when positive walls are distinctly perceptible this operation should in my opinion not be undertaken, as the result is usually negative and it may disseminate the pus and feces; I once saw a hole made with a fine Pravaz syringe in the free wall of the intestine, which soon became gangrenous, as was determined by an autopsy shortly afterward conducted.

As is well known, the diagnosis of local suppuration is to-day based upon the presence of leukocytosis. Concerning this I possess no personal experience, and no conclusive opinion has been reached by other authors.

The result of this is that it is frequently necessary to consider the other symptoms of pus. I have previously mentioned that pus may be suspected in perityphlitis when fever, either continuous or remittent, lasts for a number of days, three to five or longer; chill at the onset without prolonged fever is an uncertain symptom; it may be produced, like a fever of brief duration, by a non-purulent appendicitis and by periappendicitis. Neither are violent vomiting and severe pain positive symptoms of pus. Empyema of the appendix may be diagnosticated if, with chills and high fever of long duration, a very circumscribed, round, excessively painful tumor appears in the right lower abdominal region.

Finally, pus infection, and at that the most serious form, the putrid, is present when the so-called symptoms of sepsis or collapse become noticeable.

Occurrences of this kind are rare; in the majority of cases the fever is less intense; nevertheless, pus may be present.

With a solid tumor, and even without a recognizable tumor, pus may form in the appendix or around it, and may even be putrid, yet the temperature remain normal, and pain not be excruciating. These latter cases naturally are rare exceptions—this is the only consolation they afford.

The absence of symptoms of pus does not therefore exclude the presence of pus; but when they are absent, when the picture of appendicitis simplex, as sketched above, and with or without periappendicular symptoms, is present, it may be excluded with a great degree of likelihood; notwithstanding this, we must be on our guard.

Pus in the appendix is, moreover, a very varying condition. There is a benign pus, particularly among the cases without perforation, and with clearly defined limits; i. e., it at once causes a very hard, moderately large tumor and, after a little time, probably by rupture into the intestine, disappears; but among the non-putrid suppurations there is also a very

malignant form, usually seen in perforation, but also, although very rarely, without this. Here also the rule is operative: The malignaney, i. e., the tendency to rapid progression, is often manifest from the typieal symptoms, but it cannot be exeluded when milder symptoms only are present. That the relative position of the abscess and cecum is of importance has already been stated.

How may we recognize putrid infeetion which, in the majority of cases, indicates fecal calculus perforation? The symptoms of septic collapse, even when only indicated, must always arouse this suspicion; in a suecessful exploratory puncture a positive diagnosis can at once be made by the sense of smell. With ehill, continued high fever, very severe pain, and especially with pelvie or other severe local symptoms (marked meteorism, singultus), with albuminuria and very marked indicanuria, this must always be thought of, particularly, however, when these decided symptoms are contrasted with the absence of tumor; with mild symptoms it cannot be positively excluded, *yet it is rare.*

Comprehensively the following may be stated:

1. As a rule, vomiting, ileo-cecal pain, a moderate fever not lasting longer than three days, and very circumscribed or indistinct resistance are in favor of appendicitis simplex (inflammatory relapse of appendicitis granulosa hemorrhagica, Riedel), with non-purulent implication of the serosa. A moderately severe chill accompanies this condition; very severe pain upon palpation does not disprove it.

2. As a rule, the same symploms with protracted fever reaching to 102.2° F., and with a larger, coarse tumor indicate purulent, non-putrid, circumscribed periappendicitis; moderate meteorism occurs at the onset, but then disappears.

3. As a rule, an extensive tumor between the umbilicus and the anterior superior spine of lhe ilium with severe symptoms (chill, frequent vomiting, moderate meteorism), but from the third to the fifth day, and synchronous wilh the fall in temperature and cessation of the vomiting, a certain but limited decrease in size favors perforation of the appendix but with a tendency to solid encapsulation of the abscess which is for the most parl pulrid.

4. A sudden onsel wilh fulminant symploms, extensive painful meteorism, pelvic disturbances, and the absence of distinct resistance, awakens the fear of an insufficienl limilation of a perforalive perilonitis. Signs of so-called seplic collapse, even lhe slighlest, are, however, the positive evidence of this condition.

5. Retrocecal and relrocolic symploms (movemenls and position of the leg are lo be observed, pain in the posterior lumbar region) always favor pus, which need not, however, be putrid.

6. Chills, high fever (later declining), excessive vomiling, and very severe *pain wilh a circumscribed round lumor from the onset favor em-*

pyema. In the majority of cases this is not putrid; for this reason encapsulation of the empyema is usually firm.

7. As a rule, when several relapses occur, perforation by fecal stone will have been most severe in the first attack; if, therefore, an attack suggestive of perforation have already occurred, the relapses are usually milder; inversely, if attacks of non-perforative periappendicitis begin with an appendicitis granulosa and stenosis, usually mild at first, and gradually becoming more severe, gangrene and perforation eventually occur; the preceding attacks are then usually a certain guaranty of preceding adhesions.

8. **Exceptions to all of these rules occur, and in all directions.** *The more serious are those in which* **malignant affections set in with mild symptoms.** *The gravest condition is that of ichorous empyema and ichorous perforating abscesses with mild initial symptoms and sudden rupture into the free abdominal cavity.* **Neither occurrence is very frequent.** *Much more often is a tendency to secondary distribution gradually revealed by more severe and more distributed pain, higher fever, and more rapid pulse, vomiting and mild meteorism which will be apparent to the careful observer. Usually with secondary distribution we are dealing with mild or even moderately severe cases of progressive purulent peritonitis; almost invariably these are due to perforation from a fecal calculus. Abscesses at the medial side of the cecum are most prone to show this tendency.*

GENERAL REMARKS REGARDING THE COURSE OF PERITYPHLITIS

According to various comprehensive statistics, particularly Sahli's collective investigations, it may be stated that acute perityphlitis without operation is fatal in from 4 to 9 per cent. of all cases, and that relapses occur in about 20 per cent. of non-operative cases.

Relapses.—These relapses must occupy our attention for a moment. They occur in perforative, as well as in non-perforative perityphlitis, but even according to previous statistics they are much more rare in the former; when we consider the milder "relapses" of "appendicitis granulosa," the greater proportion of relapses in the relatively benign forms will become apparent.

The first relapse generally occurs in the first, less often in the second, year, and much more rarely thereafter.

In the perforative forms, the first attack is usually the most severe, the relapses are benign, and occasionally show a gradually decreasing severity. The earlier, however, the first relapse occurs after the first attack, the more certainly must we reckon upon its malignancy; one that occurs after a few weeks is not infrequently more serious and occasionally even fatal. These relapses may be caused by various conditions: By a propagation to the abdominal cavity of the pus which has not yet dis-

appeared (delayed progressive purulent form, malignant), to the retrocolic tissue, etc.; by the sudden occlusion of a perforation into the intestine before the pus has fully discharged; by induration and adhesions which hinder the passage of feces. Accordingly the character and the prognosis of the attack vary greatly.

Chronic appendicitis without fecal calculus, and with or without stenosis, frequently causes "relapses" of gradually increasing severity in which implication of the peritoneum is at first doubtful, but may be later more distinct; finally, pus forms and meets with adhesions previously for a long time present. But in these cases without fecal calculus, even when the first (*distinct*) attack produces periappendicular pus (usually without gangrene, therefore without perforation), the course, according to the experience of Riedel, is generally favorable: Absorption or rupture—induration for a varying time, occasionally only amounting to days—stenosis or, what is most favorable, complete obliteration of the appendix and entire recovery. In rare cases, naturally, the abscess persists, possibly with secondary complications (descent, slow rupture into an inconvenient point, for example, the bladder, venous thrombosis, etc.)—or without this, with chronic invalidism associated with pain, occasional fever, and dread of eating. Or, indistinct febrile attacks come on even when the abscess has disappeared; some time ago, in one of my cases of this kind, a torpid flat ulcer was found in the thickened appendix which was everywhere adherent. Not infrequently attacks of gradually decreasing severity are observed for years; here pus is no longer present, the adhesions and indurations gradually disappear; the nature of the difficulties produced by these conditions we shall soon refer to.

Long-continued abscesses usually give the patient no rest; however, long periods of latency (over a year) occur, exceptionally even with putrid abscesses, and under some circumstances with sudden, severe, peritoneal exacerbations.

I recently observed an instructive case of this kind.

A robust young man, 22 years of age, had two years previously a moderately severe febrile inflammation of the cecum which "healed completely"; since that time he has now and then suffered from indistinct drawing pains in the right lower abdominal region; lately he had a mild relapse, with moderate fever lasting a day, violent pain, constipation, and nausea. He was a strong, well nourished man. A tumor about twice the size of a plum, compact, round, and slightly movable, was found below McBurney's point. Slight pain upon deep pressure aroused a suspicion of pus; the conditions were otherwise negative. *Operation: Old non-putrid abscess around a perforated, otherwise markedly thickened, appendix; fecal calculus.*

If abscesses, after a prolonged duration, finally rupture into any organ, it may happen that, in the meantime, rigid cavities will have formed which, even though communication is open, do not heal; therefore the result is invalidism. But even old, closed, rigid wall cavities may form

which mechanically act as tumors; my last observation of this kind showed in a patient aged 50 a rigid tumor, round as a ball and the size of a child's head, situated between the bladder and the rectum, and causing great difficulty in defecation.

If pus has disappeared, leaving behind it permanent adhesions and induration, usually mild "relapses" occur—disturbances which come and go and scarcely deserve the name of relapse. In part these may consist of difficulty in the passage of feces, and, depending upon the seat of the induration, there may be a cecal fecal stasis which may cause secondary typhlitis stercoralis. This was probably the case in the following observation:

A man, aged 64, who, twenty years previously, had suffered from severe inflammation of the cecum, was subject from that time on, at first every year and later, up to the time of death, every few years, to relapses, the severity of which varied but generally lessened, and constantly suffered from marked constipation. Death was due to apoplexy. Autopsy: Appendix slightly adherent and obliterated, transversely over the cecum in an oblique direction a narrow, constricting thickening of the serosa (compare Fig. 20).

But adhesions of the cecum without constriction, peritoneal plaques, and adhesions of the appendix by torsion are also sufficient to cause occasional pain during peristalsis. That the latter condition may give rise to the retention of secretion and further complications (see above) by causing a stenosis of the appendix is self-evident.

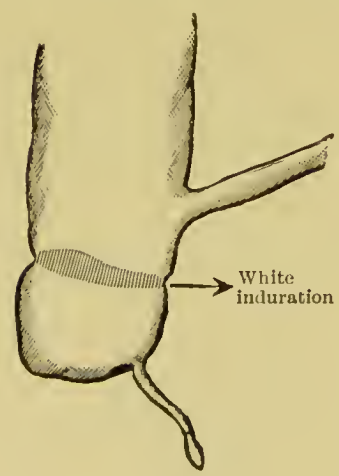

Fig. 20.

It has been previously explained, and is reiterated like a red-letter line throughout the pathology of perityphlitis, that two conditions are responsible for the majority of cases of perityphlitis: Fecal calculus and chronic exacerbations of appendicitis. Can we diagnosticate these before they produce perityphlitis? The answer to this question is scarcely satisfactory.

Fecal calculus before it produces inflammation is completely masked; of premonitory relapses of pure appendicitis which precede gangrene, and of periappendicitis, we know but little. Nevertheless, in perityphlitis we often have a history of slight drawing and tearing pains in the ileo-cecal region.

Chronic appendicitis and stenoses, on the other hand, readily produce the previously mentioned "relapses," beginning mildly and becoming more severe. In the future these will be more closely observed. For the diagnosis of perityphlitis it is highly desirable that we should in time obtain further knowledge of these prior conditions.

Peritonitis originating from *neighboring organs in the peritoneum,* or from chronic purulent circumscribed peritonitis, belongs to the realm of diseases of these organs. I shall, therefore, permit myself only a few general remarks in regard to them. The points of origin of circumscribed peritonitis are just as numerous as, or even more so than, in acute diffuse inflammation of the peritoneum. All portions of the gastrointestinal canal, the liver, and particularly the biliary passages, the pancreas, the lymph-glands, the female genitalia, the kidneys and the bladder, and finally, the actual surroundings of the abdominal cavity (the diaphragm from the pleura or from the pericardium, the ribs, the vertebral column and the pelvis) must be considered.

These conditions are partly related to specific (typhoid, dysenteric), partly to non-specific ulcerative processes, partly to infections of normal secretions (bile, urine), partly to infection of the tissues of the liver, of the genitalia, etc. Occasionally chronic processes, as echinococcus, tuberculosis, carcinoma and other neoplasms, are behind the acute process, and only manifest themselves on rupture into a hollow organ or by preceding suppuration.

Whether in these cases an acute, diffuse, and extensive or a limited peritonitis occurs, depends upon the nature of the inflammatory agent, also partly and chiefly upon the rapidity with which it reaches the peritoneum; the location of the point of rupture and whether or not this is favorable for encapsulation also plays a rôle.

Gastric ulcer sometimes produces perigastric abscesses when it slowly approaches the serosa and forms adhesions; but, even with a rapid rupture, if it is situated in the smaller curvature or upon the posterior wall of the stomach, circumscribed peritonitis below the diaphragm and subphrenic abscess may develop, because here the conditions for limitation are favorable.

Infection of the biliary passages, as a rule, at first causes mild and then severe relapses, similar to the appendicular ones, and thus tough adhesions are produced; in consequence of this perforating or non-perforating pericholecystitic suppuration is for the most part localized.

A perforating typhoid ulcer only exceptionally causes circumscribed, but usually diffuse, peritonitis; on the other hand, the dysenteric ulcer which more slowly invades the deep tissues most often produces local inflammation.

Puerperal suppurative processes of the uterus and of the adnexa are usually highly virulent and progress rapidly; here we have, as a rule, the conditions for diffuse peritonitis. Most manifold local inflammations may arise according to the virulence of the poison and the rapidity with which it enters the abdominal cavity.

There can be but few points of preference for circumscribed purulent peritonitis since it has been found in all regions of the abdominal cavity.

It is frequently noted in the pelvis, sometimes originating from the immediate surroundings, and sometimes descending from the appendix or from the colon. Another point of preference is the vault of the diaphragm: Here the intraperitoneal subphrenic abscess is usually situated, particularly if it contains gas; pyopneumothorax subphrenicus is usually to the left of the suspensory ligament of the liver, but also occasionally to the right of it, and rarely bilaterally. All of the organs bordering on the subphrenic region are implicated in the development of these abscesses: The stomach, the liver (that is, the biliary passages), the duodenum, the pancreas, the colon, the kidneys, the spleen, the ribs and vertebral column, the diaphragm from the pleura, the lungs and pericardium. The majority of abscesses originating from the biliary passages are situated below the liver; frequently they extend almost to the ileo-cecal region, and may cause great difficulty in the diagnosis; if, to aid us in our decision, an exploratory puncture be risked, pus admixed with bile, or frequently only pus, is found. Abscesses of the colon, often of obscure origin, are occasionally situated here, or they may be opposite the ascending colon and at the flexure; they are usually circumscribed and prone to be chronic.

The same is true of these abscesses as of the periappendicular form: They may show firm encapsulation and, particularly when small, may remain latent for a long time; according to their position they may rupture in any direction, through the abdominal wall into a hollow organ, especially into the intestine or into the bladder, or through the diaphragm and ultimately into the lung; they may by erosion force their way into a solid organ, as, for example, perigastric abscesses and abscesses of the biliary passages into the liver; by rupturing into the free abdominal cavity they may immediately produce a diffuse peritonitis or develop a progressive purulent inflammation. Finally, they may readily lead to pyemia, particularly by septic venous thrombosis. If the abscess is small and insignificant, situated deep and not susceptible to examination, a cryptogenetic septicopyemia may develop clinically.

To enter into further details is at this point impossible.

PROPHYLAXIS AND TREATMENT

Prophylaxis.—As to the prophylaxis of acute diffuse peritonitis, modern asepsis and antisepsis in the hands of the surgeon and gynecologist are powerful agents to prevent the development of this affection after operations upon the abdomen, in the treatment of abdominal injuries, and during labor. The situation is more complicated when we consider the numerous pathological conditions which expose the peritoneum to inflammation from its surroundings. Here it would be well if we could decide in the individual case whether the process has been so extensive as to produce inflammatory conditions or those that lead to danger of

rupture, and also whether an *invasion into the abdominal cavity is likely.* But, unfortunately, this is seldom possible.

In round ulcer of the stomach and of the duodenum and in enteric ulcer this is never the case, and even in the pancreas we are far from accuracy in clinical judgment. In gall-stone colic with fever, or even with chills, serious consequences may more readily be prevented; appendicitis and perityphlitis have just been accurately described. In incarcerated hernia, in strangulation and in invagination, surgical treatment which prevents peritonitis plays an important rôle. But upon this subject, and also the diseases of the sexual organs, we shall not now enter.

In not a few of these conditions the aid of the surgeon is needed before there is any thought of a diffuse peritoneal complication. But even where this has been previously impossible, the family physician or the internal clinician should consider himself honored by inviting the surgeon in consultation, should he be at hand and the patient permit it, and in obtaining his opinion so soon as a diffuse peritonitis seems threatening, or even before it has developed. Thereafter the case belongs to the surgeon.

If the physician, however, finds an acute diffuse peritonitis already developed, the question of treatment is directed primarily to the point of origin; whether, and how, it may be attacked is the privilege of the surgeon to decide.

In many cases, from a theoretical standpoint, we must strictly discriminate between bacterial and chemical peritonitis. I have previously stated of the latter form of peritonitis that it stands or falls with the bacterial focus of origin; therefore this should be immediately removed, when the rapid disappearance of the diffuse symptoms may be expected. Considerations of this kind will, under some circumstances, influence the surgeon as to his manner of procedure. But it is neither my object nor my function to enter into more minute details in regard to this. Practically, it is evident from the foregoing that the clinical differentiation between malignant bacterial and benign chemical peritonitis (peritoneal irritation) frequently occasions the greatest perplexity.

Operative Measures.—Therefore, according to our present standpoint of therapeutic knowledge, surgical relief is to be sought in all cases of acute diffuse peritonitis except those in which decided symptoms of severe peritoneal collapse have already appeared. Mild and moderately severe clinical pictures of this nature are, as a rule, not positive contraindications for an operation; but subjective judgment as to the patient's powers of resistance, particularly the condition of the heart and the vasomotor system, as well as the severity of the other toxic phenomena, leaves wide room for doubt; definite rules cannot be established. It must always be borne in mind that, as a rule, operation at first intensifies the collapse.

On the whole, a comparative estimate (in so far as this is possible) of the results with and without operation proves that in perforative peri-

tonitis, if operation is very early, i. e., inside of twelve, or, at most, twenty-four hours after perforation, there is slightly more, but, nevertheless, greater hope of recovery than without operation. Those cases are naturally the most favorable in which peritonitis has not yet developed, provided that up to the moment of operation there is no peritoneal collapse. If the latter occurs, the prognosis in laparotomy is exceedingly grave and, therefore, it is rejected by most surgeons.

A relatively unfavorable prognosis after operation is presented by those cases in which previously more or less localized relapses of progressive peritonitis have occurred, after which a severe diffuse picture is suddenly presented, and also in those cases in which, combined with a circumscribed peritonitis, the indistinct picture of diffuse peritoneal irritation had some time previously existed, this having abruptly changed to a severe diffuse inflammation; here, as a rule, there is a massive secretion of pus, widely distributed. These occurrences are usually preventable by proper surgical treatment of the preceding circumscribed peritonitis (see below).

The conditions are likewise unfavorable for surgical intervention in peritonitis to which a septic condition has been added, or in which this already coexisted, as is so frequently the case in the puerperal forms. Here also, for good reasons, the surgeon refuses to operate.

For the not infrequent cases where either a diffuse peritonitis is certain —its origin, however, not being quite clear—or in which the differential diagnosis between peritonitis and strangulation or the like cannot be readily made with certainty, early surgical treatment is generally advisable. It is a serious error to confound this condition with coprostasis, uremia or peritonism, although I can easily imagine isolated cases in which the physician, and also the surgeon relying upon his asepsis, might with an uncertain diagnosis assume the responsibility of deciding upon an "exploratory laparotomy."

These criticisms, which in part extend into the realm of surgery. I may be permitted to make because they indicate the cases in which the internal clinician or the house physician would make a gross error not to consult a surgeon, and because they show that the surgeon's aid is usually needed early, and that, to avert danger, laparotomy must speedily be performed.

From a purely practical standpoint, however, the conditions are such that where the surgeon may be readily obtained his opinion should be sought in *every* case in which acute diffuse peritonitis appears to be certain or likely, or even possible. This cannot be emphasized too strongly. If he can be reached only with difficulty, the internal clinician himself must weigh the possibilities and decide whether or not the aid of the surgeon is necessary.

Internal treatment must be resorted to in those cases in which operation is rejected and those in which surgical relief is either unobtainable

or cannot be secured at the right time. These are almost all lost, but none of them present an absolutely bad prognosis; this must be borne in mind. We may see most remarkable recoveries from the severest clinical picture, even of peritoneal collapse, and particularly in appendicular peritonitis; I have known the symptoms to disappear after rupture and recovery finally to take place in cases where, without rupture, the heart would have held out but a few hours longer. Such outcomes are unfortunately rare, *but they must stimulate the physician in each individual case to continue the battle to the end.*

Internal Treatment.—We possess no radical internal treatment of peritoneal suppuration, i. e., antitoxic or bactericidal, which promises success.

Two principal indications are to be met by the *internal treatment* of acute diffuse peritonitis: Absolute rest of the areas surrounding the focus of origin and also of the entire abdomen, and the maintenance of the strength of the patient as long as possible. With this, too, is the important consideration of relieving the sufferings of the patient.

The first indication calls for absolute quiet, avoidance of shock to the abdomen, therefore, above all, the prevention of vomiting, the stopping of all, or nearly all, food by the mouth, the arrest of intestinal peristalsis, psychical repose.

The necessity for absolute quiet is often confronted by the question of moving the patient to a hospital; here, under all circumstances, the individual case must be considered; the issue cannot be discussed from a general standpoint. The vomiting is at once somewhat favorably influenced by opium or morphin; if this, however, should not be the case careful lavage of the stomach with a cool fluid may be employed soon after a meal, particularly if we suspect a complicating ileus or a perforation from ulcer, and in other isolated cases in which we are under the impression that continued severe retching will be more injurious than the perturbation produced by the stomach-tube. In conclusion, I cannot advise the general employment of the stomach-tube, as some authors have done.

Food by the mouth is to be stopped at the onset; but, in the course of the affection, circumstances will not seldom compel us to return to this—naturally when improvement begins. The first food that may be administered is meat juice freshly expressed (by means of a meat press), then cool bouillon with the addition of Plasmon, Leube-Rosenthal's meat solution and the like, following this milk, cooled by ice, all to be given in teaspoonful or tablespoonful doses; diluted wine or cognac is permissible. The subsequent stage of beginning convalescence I shall pass over. Administration of water by the mouth should only be stopped in the severest cases; small pellets of ice may usually be swallowed, unless this increases the vomiting which may occur.

Administration of food by the rectum is by no means immaterial; as is well known, it may stimulate peristalsis in the entire colon. If we are forced by the failure of strength to employ this method, I advise only enemata consisting of two eggs with 1.0 sodium chlorid to each egg (sodium chlorid 20.0 to 200, 10 grams or one small teaspoonful per egg). If there be temperature, about 15 cm. of this solution is to be cautiously injected high up, or milk, perhaps with an egg and about 5 per cent. of sugar. Anything else will readily irritate.

Water is particularly valuable for conserving the strength, and much more indispensable than food. *The organism must not thirst!* According to the case and how water is borne, it may be given either by rectum or subcutaneously (in the thigh) in as large quantities as possible (in the latter case sterile physiologic salt solution of the temperature of the body, about 200 at a time, or by continuous infusion).

Of course, if the patient becomes chilly he should be made warm; that is, external heat should be applied.

To meet the indication for complete rest of the intestine, the use of opium in large doses, perhaps of morphin, immediately at the beginning of the disease, is recommended. It is true that the bowel rests in consequence of the peritonitis itself, although it is questionable whether this is invariably the case, for example, in those cases associated with diarrhea; at all events clinical observation proves that rest of the intestine is promoted by opium; the decisive significance of which is obvious, and, in comparison, the deleterious effects of its energetic use are unimportant. In this connection it is urged that it increases constipation, masks the course of the disease, and acts unfavorably upon the strength, particularly upon the heart. In diffuse peritonitis I believe these views to be without weight. Constipation in diffuse peritonitis is almost always unimportant, particularly when we consider the abstinence from food; what, however, opium may be said to conceal is not quite clear to me, since the decrease of pain and vomiting while other severe symptoms persist cannot deceive the physician, and the somnolence produced by drugs can lead to no diagnostic doubt regarding the true condition, provided, naturally, that actual intoxication is avoided; finally, as regards the unfavorable effect of the alkaloids of opium upon the heart, this, as is well known, is very slight and is readily compensated for by its favorable effect in diminishing pain and vomiting.

Treatment by opium is deleterious to a high degree when it is the resource of those who cannot decide upon an operation; here the anodyne and stupefying effect of the remedy is a source of great danger. For the critical physician, however, who objectively grasps the actual state of affairs, no such danger exists; perhaps he may prefer to let the patient bear a little pain for some time in order to win his consent to the life-saving operation! I can readily imagine such a state of affairs.

In a treatment with opium the fact must be borne in mind that the drug should be given as early as possible and in decided doses; with continued employment its paralyzing effect upon the intestines soon ceases; often in the subsequent course of the affection, and according to the condition of the patient, the treatment must be at times suspended. Therefore, at the onset a dose should be given hourly or every two hours, later every three hours, and then even more rarely, and when aggravation occurs increased doses of from 0.03–0.05 of pure opium or the extract of opium, or six to ten drops of tincture of opium are to be administered by the mouth. Vomiting once or twice (pellets of ice to be given) need not cause the suspension of this method of administration. If vomiting increases, the drug should be used by rectum (enemata or suppository) or morphin, which unfortunately does not have the same effect, by subcutaneous injection of about 0.01 with the intermediate pauses mentioned above. Naturally, when such doses are employed the symptoms of poisoning must be watched for, particularly those on the part of the sensorium and the pupils, and we must remember that the pupils contract under moderate toxic action; when it is very severe they dilate. Moderately narrow pupils do not contraindicate the continued cautious use of the drug. I have never seen an instance of the sudden cumulative effect of the remedy, which indicates that after a period of non-absorption several doses have suddenly been absorbed. It is possible that this occurrence as reported is due to confusion with peritoneal collapse and loss of consciousness; however, these remarks are not to be looked upon as a criticism of the reports just referred to.

Morphin acts upon the mind and the sensation of pain to at least the same extent as opium, upon intestinal peristalsis and vomiting less intensely; it is therefore an imperfect substitute for opium, which we are only to employ when nothing but the subcutaneous injection remains, or when we wish to effect a rapid action, as when pain is the most prominent symptom in the case.

Other substitutes are only to be considered when, on account of repeated attacks of vomiting, which are of very serious import, or the solution of adhesions, or the distribution of the generators of inflammation, trial is made of other drugs. Tincture of iodin in drop doses in a little water is an old remedy which is occasionally very successful; cocain is useless; only gastric lavage remains which in such cases is advisable; naturally but small quantities of water and very slight pressure are to be employed.

Of cardiac and vasomotor remedies in collapse, above all in *threatening* collapse, camphor and caffein, subcutaneously injected, are recommended.

In very isolated cases, particularly when the fever disappears, the pain and meteorism have become less, yet feces are not discharged, and there is marked dulness of the descending colon, the indication may arise to evacuate this organ carefully. I should not do more than use about 100

grams of olive oil by enema. The careful observation of the state of the bladder and, with a prolonged duration of the disease, the prevention of bed-sores, must always be borne in mind.

CIRCUMSCRIBED PERITONITIS, PARTICULARLY PERITYPHLITIS

We must consider also the therapeutic indications in perityphlitis. First, because it is the best guide to the points suitable for consideration, partly because it is the more frequent affection, and also because the consideration of all other forms of circumscribed peritonitis ranks in importance with the treatment of all the organs of the abdomen and their surroundings.

PROPHYLAXIS

The prophylaxis of perityphlitis up to the present time is somewhat chaotic. The apparent increase of this disease, particularly in the United States, has led to the supposition, which is perhaps most reasonable, that here hasty eating, insufficient mastication, and constipation are certain causative factors; the conclusion is obvious that persons who from a fear of appendicitis eat slowly and masticate thoroughly may thus become hypochondriacs. It is much better to relegate to general hygienic strictures all advice of this kind.

We are dealing with tangible objects when we consider fecal calculi which may certainly remain in the appendix a long time before they become dangerous, and also when we consider chronic appendicitis and its exacerbations, the "relapses" which we have already described.

Up to this time the latent fecal calculus cannot be diagnosticated, not even with the X-rays; here, therefore, is no question of prophylaxis: if it were possible to recognize it, and considering its danger and the present certainty of surgery, no doubt it would be well to remove it with its enclosure, the appendix.

Here the indication would be simple. Much more complex are the conditions in that other precursor of perityphlitis, chronic appendicitis: here the diagnosis is clear if the "relapses" are correctly considered, but it is not at all certain that mild "relapses" lead to purulent peri-appendicitis, or even to necrosis and perforation: The danger of chronic appendicitis is far less than the danger of fecal calculus. The question resolves itself into this, whether in pure appendicitis we should operate to prevent a purulent perityphlitis, and this is certainly worthy of serious reflection. We shall consider it in common with the operative treatment of perityphlitis.

TREATMENT

In the treatment of perityphlitis which has either been diagnosticated with certainty or with a great degree of probability, in general the following facts are to be weighed: Under internal treatment, i. e., without recourse to surgery, from 70 to 80 per cent. of all cases entirely recover, a number which is large but which, naturally, does not comport with our therapeutic endeavors; in the treatment of these cases, that therapy suffices which has for its Alpha and Omega the keeping of the intestines and the entire abdomen absolutely at rest. Of the cases that remain, no doubt nearly all might be cured by timely operative measures; in the majority of these cases, too, a surgical operation might be performed if the right time were chosen, and the patient were carefully examined and watched. In a small proportion of these cases, however, it is done too late, partly because, without fault of the physician, and without prodromes, there is a sudden perforation into the free abdominal cavity, but partly also because the case was an apparently benign one which gave hope of recovery by internal treatment yet it suddenly became dangerous, or because at the beginning it appeared to improve and a sudden fatal relapse occurred. These last two categories will always rest as a weight upon the conscience of the physician.

From the preceding observations it will be noticed that the most serious cases and those requiring most speedy operation are the following: 1. Perforation into the free abdominal cavity; 2. Larger abscesses which usually perforate, and are characterized by an increasing tumor and a tendency to further enlargement, a "progressive condition," noted particularly upon the inner side of the colon; 3. Empyema without marked adhesions, especially the putrid form. In contrast to these are the relatively mild conditions: Non-purulent periappendicitis; small non-perforative abscesses with solid adhesions, which terminate partly by absorption and partly by rupture in the direction of least resistance, i. e., toward the intestine; finally, mucus and sero-purulent, moderately virulent, non-putrid collections of secretion in the appendix.

These conditions very rarely lead to immediate danger; but during healing they may produce strictures and kinking, and by relapses develop a malignant character—although this is very rare.

Midway between these two categories, in regard to spontaneous cure, are the moderately large abscesses which sometimes show fine perforation and sometimes do not perforate, and in which the danger depends upon the continuance of preformed and fresh adhesions, upon the virulence of the pus, and upon the promptness of its rupture in a favorable locality. These abscesses are rarely absorbed. They form a large proportion of the total number of cases of perityphlitis.

I repeat what has already been said elsewhere in other words, in

order to recall to mind the general diagnostic knowledge regarding these conditions. The most extreme cases of this kind may nearly always be readily diagnosticated; but in the severe types, especially perforations, which may simulate mild forms, errors are not rare.

Even in these, but still more often in the numerous cases of moderate severity, it is not only difficult to decide exactly what condition is present but, much more so, what will be its course.

Let us assume that in all cases of perityphlitis we have a full comprehension of the anatomical structure, the position of the possible abscess, the condition of the appendix, the adhesions: *Even then, in the great majority of cases, it will be impossible to say that recovery will take place without surgical intervention, and, if so, whether it will be complete, or whether the knife will ultimately be necessary.* We cannot correctly estimate the resistive power of the adhesions to the mechanical influence of the pus which still has an inflammatory action, and whether this will still later be absorbed, that is, whether the lesion will heal by a timely rupture into the intestine.

Still greater, however, are the practical actual difficulties of diagnosis in regard to prognosis! What a gloomy outlook is there presented in regard to a future improvement in our diagnosis, particularly as a foundation for treatment! Nevertheless, we may say to-day that in by far the majority of cases of perityphlitis the therapeutic indications are so palpable that proper treatment will lead to a favorable termination.

The first question in treatment, which must be answered as soon as possible, is this: Must we operate immediately and during the attack? Most internal clinicians and surgeons will answer that we should operate in the following clinical forms:

1. In the acutest cases of perforative peritonitis without tumor, and here as speedily as possible—every quarter of an hour is of importance; those cases of well developed peritoneal collapse in which an operation can no longer be borne must be excluded; it is obvious that these furnish a fruitful field for individual judgment.

2. With secondary distribution of inflammation to the remaining areas of the abdominal cavity from an already existing perityphlitic abscess; this process, however, should be averted by a timely early operation (in regard to this, see No. 3).

3. In a large, distinct, fluctuating abscess, as well as in all acute painful perityphlitic tumors, which, either by a tendency to enlargement or by a fever which increases for several days, or by a rise in temperature which is continued for more than five days, or by a fresh exacerbation after a previous improvement, foreshadow the danger of rapid progression. Here it is our duty by every possible means to prevent the extension of the peritonitis, or even peritoneal collapse.

4. When a very painful, circumscribed, round tumor, appearing with fulminant symptoms, indicates a putrid empyema of the appendix.

In my opinion, and according to the present status of our science, in these cases there can be no doubt as to the indication for operation.

Neither, after an attack has passed, can there be the least doubt in regard to the necessity of operation in the following cases:

1. When a recognizable abscess, even without irritation, persists for weeks.

2. When, after an attack without operation, debilitating relapses occur, and particularly when the relapses show an increasing severity.

3. When there are permanent adhesions and indurations which disturb the appetite, the digestion, and motion; when these cannot be relieved in other ways (see below), and prevent recovery; and when they manifest themselves either by pain and slight inflammatory relapses or by fecal stasis.

These are, as I believe, the *absolute* indications for operation; what now remains, and this should be borne in mind, applies to the majority of cases of perityphlitis. These, as a rule, make a good recovery without operation, *but are nevertheless subject to some dangers.* These dangers are of various kinds: First, are those which arise from an error in diagnosis, from larval perforation with non-recognizable pus, etc.; also when, after a correct diagnosis—for example, in a well encapsulated abscess with a moderately severe appendicitis—a *change* occurs which could not have been foreseen (rupture into the abdominal cavity, sudden necrosis of the appendix, venous thrombosis, etc.).

If we wish to protect ourselves from these eventualities we necessarily face the question: Is it not well to operate in all cases, even in the mildest ones? Should we not even terminate the paroxysmal exacerbations of chronic appendicitis by extirpation of the organ? The surgeon, who usually sees only severe cases, and among these many with a severe relapse after a first mild attack, in surprising contrast to the benign onset, is confronted with this question which must be answered according to his skill, his temperament, and the results which he has obtained. Many a one, however, later becomes conservative.

In the hands of the *skilful* surgeon the immediate dangers of operation, contrasted with the general dangers of perityphlitis calculated purely by percentage, are decidedly in favor of the patient; however, here we are dealing with the personal methods of individual surgeons, and the most skilful are not always at hand. *This must be borne in mind by those who write regarding these conditions.* Besides, we must consider the not infrequent sequelæ of the operation, in spite of the fact that modern surgery does everything possible to prevent them; we mean hernia of the abdominal wall from diastasis of the cicatrix, and weakening of the musculature by degenerative atrophy in consequence of the section of motor

nerves. These conditions are occasionally of vital importance, especially in the female during labor. To this must be added the instinctive repugnance of man to mutilation, which in a mild illness makes it difficult for him to consent to operation for an affection which is usually only slightly dangerous.

The relegation of all forms of the disease to the knife of the surgeon will, therefore, I am firmly convinced, never be possible; on the contrary, on account of the rare dangers which, however, can never be completely excluded in the individual case in the milder forms of the affection, the question arises in how far is operation necessary.

On this point opinions are far asunder, which is only to be expected. The perplexity which assails us in an exact discrimination of the cases is very great; aside from a certain tenacious adherence to the old, which to some extent still shows itself in internal medicine, and aside from the very prevalent error of the surgical author in considering as the normal whatever he is able to do for himself, each position has its justification.

The question to be put in the "intermediary" cases is a double one: 1. Which cases are to be operated upon during the attack? 2. Which are to be operated upon after the attack? In regard to the first, many authors are of the opinion that operation is to be immediately undertaken when pus is present or when it is likely to be present. Here we must face the previously mentioned diagnostic difficulties based on the general assumption that pus always accompanies acute, painful, febrile tumors, a position that can no longer be maintained; for pus may be absent when we have quite high fever, severe vomiting, and a distinct, painful tumor. Besides, the catchword "pus" by no means embraces everything; the dangerous character of a periappendicular pus focus varies according to the position and the length of existence of the adhesions, according to whether or not perforation takes place, whether or not it is putrid—and not only the local but also the general symptoms are of significance; they may manifest severer, or milder, conditions than those present. Finally, pus may be absent in one "relapse" and appear in the next one.

I advise, in general, in those cases in which pus may be assumed and in which there is no solidity of the tumor, when it decreases in size at the latest about the third day, with a retardation of the fever and improvement in the general condition and pulse, all of which point to good encapsulation—in such cases, a consultation with a surgeon and, if possible, the consideration of an operation. When the previously mentioned symptoms of encapsulation appear there will rarely be an aggravation of the condition, and, should this occur, it will almost always show itself by certain symptoms (enlargement, meteorism with moderate pain upon pressure, renewed rise in pulse and temperature, tendency to vomiting, dysuria, etc.), so that operation may still be undertaken in

good time. For this, however, it is necessary **carefully to watch** the patient, and this, in fact, should be done as conscientiously as possible; where, however, it is impossible, the list of operative cases will be enlarged. *In these intermediate cases the question whether or not the patient can be watched has a strong bearing upon the question whether or not we shall operate in the attack.*

In many of these cases, if we do not operate during the attack, the question of operation may again arise in a few weeks, and invariably if there is a suspicion of persisting pus.

Operation after an attack has advantages and disadvantages in contrast to those of operation during an attack. I shall not enter upon the pros and cons, as this is a purely surgical question. Whoever rejects operation during the attack must naturally take account of the risks. Here, too, watching the patient is specially important.

Neither can I answer the question as to the method to be chosen, whether immediate removal of all the pathologic conditions, or the opening of the abscess and the subsequent extirpation of the appendix.

It is not my function to discuss the surgical indications in slowly progressing cases with the appearance of new pus foci in the abdominal cavity, in the belly wall, or in the pelvis behind the colon (examining the lumbar region!), in the subphrenic tissues, or in the pleura. But it is the duty of the physician on the slightest sign of complication of this kind, for which search should be made in all cases, to call in the surgeon. Prior to this, however, these cases have usually come under surgical treatment.

Non-operative Treatment.—I have previously stated that the Alpha and Omega of treatment in those cases of perityphlitis and also of appendicitis itself in which an operation is, or appears to be, unnecessary, or is not permissible, is to keep the intestine and the whole abdomen quiet.

The indications are primarily the same as in diffuse peritonitis: Rest (great care in transportation to a hospital), no administration of nourishment by the mouth at the onset or even later, nor, if possible, as long as inflammatory symptoms continue; if these retard, or the condition of the patient shows that the strength must be increased at any cost, the same dietary mentioned upon page 561 should be employed. The intake of water by mouth must be moderate, always small quantities only at a time, and perhaps only ice. At the onset food should never be given by rectum; nor should enemata then be given to empty the rectum; nevertheless, in protracted cases it may be necessary to risk nutritive enemata when that mentioned upon page 562 may be used. In such cases the subcutaneous injection of water may also be necessary.

The application of ice or cool or lukewarm Priessnitz compresses may depend upon the wishes of the patient. At the onset hot cataplasms must be strenuously forbidden.

Here also opium is the sovereign remedy; it must be employed in *all* cases of appendicitis and perityphlitis, provided it does not induce vomiting; in this case suppositories are to be resorted to, and only when these are not well borne should morphin, which does not act so favorably, be used as advised upon page 563.

In the old so-called typhlitis stercoralis it was found necessary to vindicate the position of opium, and this is the case even to-day. The same deleterious effects are alleged as in the case of acute peritonitis, and it is maintained that by increasing the stagnation of feces it induces a relapse in appendicitis (Riedel). Particularly is this true in perityphlitis, which frequently shows a retardation of all the symptoms after the first fecal evacuation, and invariably leads to the assumption, similarly as in stercoral typhlitis, that any evacuation of the intestine as such acts favorably, and that stagnation of the feces therefore acts unfavorably, so that we dare not increase the latter condition.

It must be admitted that those who, in milder cases of acute perityphlitic tumor (therefore, those in whom a cecal stasis of feces was previously looked upon as the cause of the disease), risk a decided dose of castor oil occasionally see all the symptoms disappear when the oil has had its full effect; in such cases it is questionable whether we are not dealing with a relapse of a chronic appendicitis (without periappendicitis!) and secondary fecal tumor from intestinal paralysis, or whether the appendicitis was not favorably influenced by the evacuation of the large intestine. I can readily imagine that the future will see a purgative treatment of chronic appendicitis and an energetic purgative treatment for acute relapses, *provided that, in the individual case, the presence of a fecal calculus in the appendix and the presence of inflammatory products, etc., of the appendix may be excluded with absolute certainty, and also that we may exclude a beginning invasion of the serosa; to-day we have not advanced so far!*

In every case, however, of fecal calculus, or when, without fecal calculus, products of inflammation appear upon the surface of the serosa, or when they are extending to this tissue, or have already formed adhesions, the stimulation of peristalsis is a grave danger, and absolute rest of the intestine is the principal indication.

And, since it is as yet impossible to exclude these cases with certainty in acute disease accompanied by pain in the right iliac fossa, purgative treatment in cases of this kind should not be resorted to. According to the present status of diagnosis, we must reject this form of treatment, not only by the mouth but, with very recent inflammation, for even a longer time by rectum.

Opium, on the other hand, is even more valuable here than in diffuse peritonitis; for the salutary intestinal paralysis due to inflammation in circumscribed perityphlitis implicates only the nearest and most adjacent

parts: The cecum, a portion of the colon, perhaps the lowest portion of the ileum; it is, however, necessary to prevent peristalsis in wide areas, to increase the localization of the poison, and to moderate the propulsion of the contents of the small intestine toward the lowest portion of the ileum and cecum. This latter point appears to me to call for a little reflection.

Therefore, in every acute case, as in the case of acute peritonitis, the treatment should be opium by the mouth in large doses, abstention from food, and absolute bodily rest in so far as this admits of the transportation of the patient; with repeated vomiting, suppositories, possibly morphin subcutaneously.

When there is stubborn and violent vomiting, particularly, however, when there are symptoms of intestinal obstruction, subcutaneous injections of morphin are indicated, as well as when very severe pains are present which necessitate speedy relief; here this remedy may also be combined with opium.

In the opium treatment, however, it must be borne in mind that prolonged, continued doses of opium rapidly lose their effect upon intestinal peristalsis, as is usually evident in the treatment of chronic diarrhea; therefore, it is advisable that after the administration of two doses, smaller doses should rapidly follow or the treatment be interrupted if the circumstances at all warrant this (fever, vomiting, pain, meteorism, decrease in the size of the tumor); with the slightest aggravation the dose should be immediately increased. In severe cases in which operation is declined, or in which it is impossible, this drug under some circumstances may also be given for weeks with remissions and intermissions, and in these patients we will have an opportunity of observing the highly ameliorating and localizing effect of the remedy.

In severe cases, the periodic administration of mild narcotics, even when the pupils are moderately contracted, is permissible, and is recommended; naturally, in such cases there must be careful and experienced watching continuously. Where this is impossible the doses must be smaller.

The most serious misuse of this remedy should, however, be strenuously avoided; it consists in this, that physician and patient are content with the amelioration of pain instead of deciding upon an absolutely necessary operation!

In regard to local derivatives in the acute stage, leeches alone can be considered, as is also stated by Nothnagel; I have seen amelioration from their use (which, however, may also be induced by opium or morphin), and perhaps lessening of the vomiting, but scarcely more than this. With the previously mentioned indications they may occasionally be applied in very robust individuals, at least four at a time.

If the perityphlitis ameliorates, the local application of cold should

be followed by moist heat, and this, if possible, should be continuously used until the resistance disappears; food may be very cautiously administered according to the indications upon page 561. Drugs had better be avoided. It is important in this stage that patient and physician wait for the first fecal movement, in case this has not yet appeared.

I saw two serious relapses which occurred after the disappearance of fever for several days and with an almost painless limited tumor and otherwise entirely normal abdomen, after the cautious injection of a small, lukewarm enema, and since then I leave the rectum alone as long as possible, all the more so as I cannot remember having seen a relapse which was due to constipation. Now and then accumulations of feces in the colon are uncomfortable for the patient, although here a psychical factor, i. e., the knowledge that the feces have not been voided for such and such a length of time, is coöperative; but this, too, is exceptional; constipation of eight days and longer is usually well borne. In the case of tenesmus it is self-evident that the patient must be cautioned against employing the abdominal press, but this caution is often neglected. Following the first spontaneous and very copious fecal evacuation I have observed renewed sensitiveness upon pressure and fever. Not a few patients report a "wound" sensation upon the right side low down. This indicates the need of care. In case of necessity, after the first evacuations have occurred, emulsions of oil or castor oil may later be given by rectum.

With a tumor still decreasing in size, with continued fomentations, the diet must be strict (gruels, eggs, meat juice, beef tea, cooked cereals); the patient must be kept in bed until all symptoms have disappeared. If absorption of the tumor is delayed, if, moreover, there is not the slightest indication of pus after two, three or four weeks, the time comes when the patient can no longer be kept in bed, and more solid food cannot be denied him. At all events, he must not leave his bed until spontaneous pains have disappeared, until the freest movement of the right leg is painless, and the pain upon pressure, except an indistinct, dull sensation, has disappeared; then an abdominal bandage (which should also be worn while in bed between the fomentations) must be worn; it should firmly support the parts and protect them; the patient should remain in the recumbent posture as much as possible, particularly after meals. The bowels must be carefully regulated, above all, by the unsurpassed enemata of oil. There should be no sudden movements, no bending, etc., for the first period.

Subsequently and for a long time the physician should watch the abdomen and the fecal movements. If the latter are disturbed he must ascertain whether intestinal torpidity, mechanical obstructions by induration and adhesions, or new inflammatory irritation is the reason. In the latter case absolute rest, abstention from food, and, perhaps, according to the intensity and duration of the symptoms, opium, are indicated, but

by no means a purgative. If intestinal torpidity or obstructions due to induration, etc., are present, mild purgative treatment is advisable, but in the latter case we must be careful, above all, when infectious perityphlitic residues cannot be excluded with certainty. Under these circumstances it is obvious that purgative enemata are often to be preferred to drugs by the mouth.

The treatment of persistent indurations with various baths, above all, with mud baths, fango, poultices, etc., is, as is well known, often effective. Satisfactory results are doubtless now and then seen from massage, but usually it is productive only of harm, and as, up to the present time, it is impossible to foreknow the cases in which it will be useful and certainly do no harm, the method is too risky and should be rejected; still more is this the case with horseback riding, bicycling, etc., which are now and then advised.

For the rest, many conditions which are here considered will also be discussed in the description of chronic peritonitis.

II. CHRONIC PERITYPHLITIS

By J. BOAS, Berlin

As is the case with acute perityphlitis, chronic inflammations of the vermiform process have multiplied with the changes produced in this realm of disease by surgical intervention. By its agency our formerly vague conceptions of the nature and the course of inflammatory processes have assumed definite shape, and are based upon exact knowledge. Laparotomy has solved numerous problems which, up to very recent times, still clouded the teaching of perityphlitis, and solved them much more completely than did necropsy.

Internal medicine with its greater conservatism followed the paths marked out by laparotomy only slowly and cautiously. But the impression produced by surgical successes was soon profoundly felt. Gradually it was conceded that in chronic inflammations of the cecum, even more than in the acute form, internal remedies were ineffectual. Convinced by surgical achievements, although at first incredulous, internal medicine retreated from its original position.

But this complete change of view furnished to internal medicine new problems to be solved. If perityphlitis is not to become a mere byplay in the operative technic of many surgical enthusiasts, as to-day appears to be the case, although only in isolated instances, internal medicine must closely unite with surgery in weighing the indications and contraindications of operative and internal treatment, and thus satisfactorily explain their many contrasts. Moreover, it is the duty of internal clini-

cians to fill out the numerous gaps which still appear in the teaching of acute and of chronic perityphlitis.

In the treatment of neither form has the final word been spoken. In acute perityphlitis especially, the revision of our present therapeutic method is all the more necessary because here surgical measures do not always avert the dangers which threaten life.

From this it is evident that in the teaching of perityphlitis there is not, as is often stated, a boundary dispute between internal medicine and surgery but, on the contrary, an honorable contest, worthy of the zeal of the most noble in both branches of medicine, to attain the same end: To preserve by the best possible means the life of the patient who is in danger.

From this point of view I shall enter upon the discussion of chronic perityphlitis, which with its frequency and obduracy so often falls within the domain of the practising physician.

Before describing the clinical picture of chronic perityphlitis, a few remarks may be in order regarding the nomenclature of the disease, particularly since, by an improper designation, two fundamentally different forms of the disease are often confounded with each other.

The Americans and the English have long differentiated between two forms of chronic appendicitis: *Relapsing* and *recurrent appendicitis.* The latter term they apply to a recurrence of an acute attack, while by relapsing appendicitis they mean a chronic form developing insidiously. In French literature these forms are divided into *appendicite à rechutes* and *appendicite à recidives* (Talamon).

I recently proposed [1] the designation *residual perityphlitis* for that form which continues after an acute attack, that is, the recurring form, and for the second form which runs a chronic course the term chronic recurrent perityphlitis.

How important is the differentiation of these forms of perityphlitis will be seen from the following:

ETIOLOGY OF CHRONIC PERITYPHLITIS

What is the origin of chronic perityphlitis?

Residual perityphlitis develops, as remarked above, upon the basis of an acute attack. It may be stated, as a law, that anatomically every acute attack leaves residua. But these need not result in functional disturbance. In some cases, however, residua may be permanent, and continuously produce more or less severe symptoms.

In the chronic relapsing form of perityphlitis the conditions are re-

[1] *Boas,* "Ueber die Diagnose und Therapie der chronischen Perityphlitis." *Deutsche med. Wochenschr.,* 1905, Nr. 27.

versed. Here the preceding chronic intestinal catarrh forms the chief and most frequent etiologic factor.

These intestinal catarrhs may vary in character. They may appear as an ordinary colitis mucosa or colitis membranacea, or they may be ushered in with the symptoms of chronic catarrhal diarrhea.

Habitual constipation, spastic as well as atonic, may occasionally be the precursor of chronic perityphlitis. But in my experience actual colitis is by far its most frequent cause.

It is well known that, in a small number of cases, the intestinal canal may function in a perfectly normal manner prior to the development of chronic inflammation of the appendix.

Besides chronic intestinal catarrh, in the female the uterine adnexa upon the right side play an extraordinarily important rôle;[1] disease of these adnexa may be communicated to the vermiform process, or, *vice versa,* the appendicular process may gradually spread to the right tube and the right ovary. How often disease of the appendix and a gynecological affection may simultaneously occur is shown by the fact that among 75 gynecological cases Hermes found in no less than 40, therefore in 53.3 per cent., demonstrable changes in the appendix.

Furthermore, as has long been known of acute perityphlitis, in the chronic forms traumatic influences may be of etiologic significance, and cause so-called latent inflammation to become manifest or irritative conditions suddenly to exacerbate into acute attacks. In the chronic relapsing form not only acute and powerful but continuous and weaker mechanical irritation may be a factor.

ANATOMICAL AND HISTOLOGICAL CHANGES IN CHRONIC INFLAMMATION OF THE APPENDIX

Laparotomy has permitted us a deep insight into the changes which the diseased appendix and its surroundings undergo in acute and chronic inflammation.

According to the most recent investigations of Aschoff, the inflammatory process in *acute* cases begins in the crypts of the mucous membrane. The pathogenic microörganisms which here collect in consequence of toxic action first damage the epithelium of the mucous membrane. By this effect of the toxin, or by the subsequent entrance of bacteria, a circumscribed softening of the muscularis takes place while macroscopically no changes are visible in the serosa or in the mucosa. Either these altera-

[1] Compare *O. Hermes,* "Ueber einige Beziehungen der Appendicitis zu Erkrankungen der weiblichen Genitalorgane." *Deutsche Zeitschr. f. Chir.,* Bd. L. Also, "Erfahrungen über Veränderungen des Wurmfortsatzes bei gynäkologischen Erkrankungen." *Ibid.,* Bd. LVIII; further, *Th. Landau,* "Wurmfortsatzentzündung und Frauenleiden." Berlin, 1904, Aug. Hirschwald.

tions disappear or a small intramuscular abscess forms which ruptures into the peritoneum.

During this perforation the mucosa is still intact. If a fecal calculus is accidentally present in this area, it occasionally vaults the still intact mucous membrane below it. The inflammation extends more deeply into the mucosa. Diphtheritic coating and softening of the mucosa appear. Through the ruptured mucosa the fecal calculus finds its way. These fecal calculi in themselves are benign. Only when the vermiform appendix is simultaneously affected does the stone assist the process by localizing the inflammation, thus promoting a severe course and retarding the process of healing.

The estimation of anatomical and histological changes in chronic relapsing inflammation of the appendix is much more difficult.

Long ago v. Renvers and Finkelstein pointed out that anatomical and histological changes in the appendix are found at the necropsy of individuals who, *intra vitam,* never showed any disturbance of this organ. In a remarkably thorough study Tuffier and Jeanne [1] lately determined the numerous abnormalities of the vermiform process under normal conditions in regard to size, position, and form. I think it advisable to enumerate some of the most important findings of this investigation.

The position of the normal vermiform process varies from 2½ to 20 cm. and more; it is usually from 7 to 11 cm. in length. From its insertion at the apex the diameter is generally uniform. If the vermiform process is incised longitudinally, and the breadth of the organ is measured, the width is found to be from 4 to 17 mm.; upon the average it is from 10 to 13 mm. Yet there are many normal cases in which the organ is club-shaped, i. e., the diameter increases from its insertion to its free end so that the breadth may be from 3 to 6 mm.; more frequently the inverse is found, and the caliber then gradually decreases toward the free end, perhaps to such an extent that the last 2 or 3 cm. of the organ are thready and resemble thin silk, the passage finally being reduced to almost nothing.

The opening of the vermiform process into the cecum varies in different individuals; it is circular and funnel-shaped.

The position of the vermiform appendix is sometimes

below and parallel to the ileum	= sub-ileal;
anterior to the ileum	= pre-ileal;
posterior to the ileum	= retro-ileal;
below the cecum	= sub-cecal;
posterior to the cecum	= retro-cecal;

[1] *Tuffier* and *Jeanne*, "Étude anatomique sur l'Appendice et la région iléocecale, basée sur 180 necropsies." *Revue de gynécologie*, 1899, p. 235, quoted from *Th. Landau*, "Wurmfortsatzentzündung und Frauenleiden." Berlin, 1904, p. 4 u. f.

at the anterior surface covered by the cecum = internal retro-cecal;
anterior to the cecum = pre-cecal;
posterior to the ascending colon = retro-cecal;
beside the colon = para-cecal.

Sometimes the vermiform process is found in the pelvis, sometimes above it.

As to the form of the vermiform process it is usually rectilinear; often, however, it is slightly curved or bent, sometimes sharply kinked to form a right angle, or it may be hook-shaped or semilunar.

Its relation to neighboring organs also varies greatly, and naturally depends upon the manner of insertion but also to some extent upon the movable cecum and the position of the vermiform process itself. It may extend to the liver, or may be anterior to a displaced kidney; sometimes it may reach to the rectum or the sigmoid flexure, at other times nearly to the bladder.

The contents of the normal appendix differ both in quantity and quality; often a fluid or soft, doughy mass is found, sometimes small, hard, fecal particles, or it may be empty.

In regard to the so-called Gerlach valve, the recent interesting investigations of v. Hansemann [1] show that its power of closure bears an intimate relation to the presence or absence of fecal concrements. The same is to a certain extent true of regurgitation into the cecum.

The anatomical changes in chronic relapsing perityphlitis are manifold, and, corresponding to the nature of the case, they are relatively slight. According to Federmann,[2] whose description we shall follow, the mucous membrane is more or less thickened. In the mucosa we find a plentiful accumulation of round cells or connective tissue proliferation with swelling, bulging and polypoid excrescences (appendicitis polyposa). In other cases the decrease of the glandular tissue leads to atrophy or may cause partial or total obliteration of the lumen.

In other instances the appendix is more or less thickened, rigid, or erect, as Talamon expresses it, often abnormally adherent or firmly agglutinated to its surroundings. Frequently, however, it is absolutely free. The lumen of the rigid structure gapes, and is filled with a smeary, mucoid, or hemorrhagico-purulent mass admixed with portions of feces or fecal concrements of disagreeable odor. The mucous membrane is thickened, moderately tumid, injected, and in many cases is covered with ulcers. The muscular layer is distributed partly by inflammatory infiltration, partly by true labor hypertrophy. The surroundings of the process are

[1] *v. Hansemann, Mittheilungen aus den Grenzgebieten der inneren Medicin und Chirurgie*, XII, p. 514, u. f.

[2] *Federmann*, in Sonnenburg, "Pathologie und Therapie der Perityphlitis." 5. Aufl., Leipzig, 1905, p. 48.

also implicated. The appendix is more or less adherent to the cecum, to the neighboring intestinal coils, and to the parietal portion of the peritoneum, and this leads to stasis of secretion, and the accumulation of mucus and fecal constituents.

If obstruction occurs during a time in which the cavity of the vermiform process is empty, and if no virulent microbes are present in the obstructed area, inflammatory processes do not arise but the cavity becomes more or less distended, and so-called dropsy of the vermiform process sets in. The glandular apparatus undergoes a gradual retrogressive change, the epithelium is desquamated, the mucous membrane loses its original character, and all of its constituents undergo hypertrophy. The extent of the dropsy may attain the size of a fist. If during the time in which adhesions are forming many virulent bacteria are present, empyema of the vermiform process results, or a dropsy empyema may subsequently arise from secondary infection.

THE CLINICAL PICTURE OF CHRONIC PERITYPHLITIS

I. RESIDUAL PERITYPHLITIS

We have already stated that every acute attack of perityphlitis leaves traces in the vermiform appendix or in its surroundings. Nevertheless in a clinical sense the affection may be regarded as having run its course provided there are no subsequent attacks or other sequelæ. Such a cure, however, does not occur in the majority of cases, and we must always count upon the possibility and even likelihood of a relapse soon or late. Daily experience teaches us that the greater the lapse of time after the first attack the less the danger of a relapse. This, however, is by no means absolutely true, for under some circumstances relapses more or less severe may develop years after the first attack. Some authors incline to the opinion that this is not a true relapse but a new attack; yet from the fact that small pus foci may remain for years encapsulated in the surroundings of the appendix as well as that numerous mechanical changes in the appendix (obliteration, inflammatory thickening, adhesion, exudates, dropsy, empyema) follow an acute attack, the view of an acute relapse seems more likely.

A relapse following an acute attack may develop in three different ways, as follows:

1. Acute attacks occur after certain intervals and these may differ entirely from the first in severity and intensity. It must here be emphasized that a mild, primary attack may be followed by very severe and rapidly fatal, secondary relapses. To this category belongs the following case which I observed a few years ago:

S. B., a merchant, aged 24, was attacked in the spring of 1902 with mild perityphlitis and moderate fever. Against my advice the patient made a long foot tour

of Switzerland in the following summer. Upon the 3rd of September, following an angina follicularis, possibly also after an error in diet, severe pain appeared in the abdomen, especially in the ileo-cecal region. The temperature rose to 101.3° F.

When I saw the patient the next day with the family physician the general condition was good. The temperature was 101.1° F., the pulse 92 and of good tension; he had slept well during the night. Above Poupart's ligament on the right there was a moderately dull, tympanitic zone which was sensitive to pressure.

Upon the 5th of September, 1902, there was jaundice and repeated vomiting occurred, *the vomitus being distinctly hemorrhagic* (guaiac test positive). Slight meteorism was present. The pulse was 96, the temperature 102° F.; the hepatic region was insensitive to pressure, and the same condition was present in the ileo-cecal region as on the previous day. Considering the severity of the case operation upon the same day was advised. The surgeon was E. Hahn. Finding: Gangrenous appendix with suppuration into the surroundings. Collapse and death upon the 7th of September, 1902.

Here we were dealing with one of those quite rare cases designated by Dieulafoy [1] as *vomito negro.*

On the other hand, after an exceedingly severe case, one or several mild relapses may follow.

The assumption is obvious that relapses are the immediate result of mechanical conditions (trauma, too great bodily exertion, acute indigestion, etc.), but there are also cases in which the most scrupulous care in questioning will elicit no clue to the cause.

2. The sequelæ of an acute attack are of exceedingly chronic character. Among them are chronic exudates or indurations, adhesions about the vermiform process, empyema or dropsy, thickening or kinking of the appendix, and other similar affections of the appendix or in its surroundings.

The clinical course in this group of cases is, as a rule, the same as that of chronic relapsing perityphlitis (see below).

3. The clinical picture of chronic relapsing perityphlitis may already exist but suddenly more or less severe acute symptoms develop. These are probably to be attributed to purulent residues or ulcers upon the mucous membrane of the appendix, but even sudden perforation may be the cause. The case now to be described, which at the same time proves that even after repeated rupture through the intestine with discharge of pus recovery may occur, is an illustration of this form.

Miss L., from W., aged 22, whose parents are living and well, and who states that she has always been healthy.

Toward the end of February, 1903, she was attacked by pain in the region of the cecum with vomiting, constipation, and a temperature of 102.2° F. An abscess formed, and after ten days an incision was made from the rectum and a tampon introduced. On incision a large amount of pus was discharged. Following this the patient remained in bed for three weeks but did not recuperate; she emaciated so greatly that in the spring she was sent to Pyrmont. Here, eight days later, she

[1] *Dieulafoy, Bullet. de l'académie de médec.*, 1901, No. 6.

was again attacked by symptoms of perityphlitis, and for the second time pus was discharged through the rectum. Notwithstanding this the pain persisted for some time, and the patient was confined to bed for eight weeks. She gradually recovered, but continued very weak. Upon September 7th, 1903, she entered a sanatorium.

The patient did not complain of pain, her bowels moved regularly, and she was clinically treated merely to improve her nutrition; she was carefully watched.

Status Præsens: The patient is small, undeveloped, and emaciated, with scanty musculature and a delicate frame. No edema, no eruption, no enlargement of glands. Patella reflex increased, pupillary reflex normal. Lungs normal.

Heart: Dulness normal; the second sound markedly accentuated but pure.

Abdomen: Only a small circumscribed area midway between the navel and the spinous process of the right ilium shows slight sensitiveness to pressure. No abnormal dulness.

Urine contains neither albumin nor sugar.

Bowel movement well formed with no admixture of pus or mucus.

Weight 43.5 kilograms.

Treatment: Alcohol compresses over the region of the appendix, rest in bed, stimulating diet.

Under this treatment there was decided improvement and a marked increase in weight. The patient left her bed for the first time on the 3rd of October, 1903. Her weight was then 47 kilograms.

About half-past seven in the evening the patient suddenly complained of severe pain in the region of the cecum. The temperature was 98.2° F. Cold compresses were applied, and suppositories of codein and belladonna were given; at midnight, on account of increasing pain, 0.01 of morphin hypodermically. The temperature was normal.

October 4th, 1903. The pains had been less severe after the use of morphin, but in the morning they increased. Treatment: Morphin 0.01 subcutaneously and ice-bags. In the ileo-cecal region there was an area of resistance about the size of a plate and markedly sensitive to pressure. The note upon percussion was very dull. The temperature in the morning was 99.1° F., in the evening 100.6° F. An ice-bag was ordered, also fluid diet, and 15 drops of tincture of opium every three hours.

A consultation with Prof. Körte was held on the evening of the same day. On considering the danger of an immediate operation and the fact that the temperature was but slightly elevated, the pulse 90 and strong, the decrease of the pain and the absence of peritoneal symptoms, it was decided to postpone operation and to wait until the acute symptoms disappeared.

October 5th, 1903. In the morning the condition was somewhat better; the temperature was 98.8° F., the pulse 108. There was still sensitiveness upon pressure, especially in the region of McBurney's point, and, although to a lesser extent, to the left of the navel. The resistance in the region of the cecum was apparently somewhat less, the percussion note rather dull, but normal in other areas. There was no singultus, no vomiting. The treatment was continued.

Afternoon: Condition less good; pulse 120, temperature 100.2° F. Toward evening the general condition worse, pulse 128, temperature 100.6° F. The pains in the region of the cecum and also upon the left side had become very severe. Flatus was passed twice; there was no eructation, no vomiting. On account of the danger of perforation Prof. Körte operated at 10 o'clock at night, the patient being under chloroform anesthesia.

The posterior surface of the colon was covered with purulent coating, the remaining serosa of the intestine being smooth and glistening. The vermiform process was found deep down below the colon and was elevated. A point from which pus was

discharged could be recognized. The appendix was about 6 cm. in length, and greatly swollen; it was extirpated in the usual manner.

Course: Without complications.

The patient was discharged cured upon the 14th of November, 1903, weighing 50.5 kilograms.

II. CHRONIC RELAPSING PERITYPHLITIS

In contrast with residual perityphlitis, chronic relapsing perityphlitis is of much milder and more benign type. Yet it is often characterized by such an enormous number of individual points, variations and complications as to make it an extraordinarily interesting affection.

In the chronic relapsing form of perityphlitis two distinctly different varieties may be diagnosticated: First, that running its course without an acute attack, or, more correctly, acute exacerbation, and secondly, that in which the chronic process is interrupted by mild attacks now and then occurring. In very rare cases (Rosenheim [1]) of these forms perforation is said to occur suddenly.

However, it is doubtful whether in these instances we are not dealing with abortive cases of residual perityphlitis. At all events a favorable and benign course in chronic relapsing perityphlitis is so much the more frequent that practically we need not consider the previously mentioned serious eventualities.

In a study of chronic relapsing perityphlitis we meet with a number of forms whose clinical symptoms are so well characterized that their recognition is of great practical importance.

We shall begin with *latent perityphlitis.*

(a) **Latent Perityphlitis.**—Although this form is not technically the subject of professional treatment, we particularly desire to discuss it, primarily because it plays an important rôle in the prophylaxis of perityphlitis. These cases may occur either in persons who years before passed through a positive attack of perityphlitis or in those who have never shown any of its symptoms. The patients who consult us for other disturbances present absolutely no symptoms referable to the ileo-cecal region, but careful and repeated examinations reveal a very characteristic sensitiveness to pressure in this area. Among 106 cases of chronic perityphlitis that I observed a year ago I noted this latent perityphlitis in 23. It is true the differentiation of such cases is difficult for, according to the reports of Keith,[2] McBurney's point is normally sensitive to pressure, but, like Lennander (loc. cit.), I emphatically deny this.

It may be admitted that McBurney's point is tender on exaggerated pressure, but a comparison of this sensitiveness with that caused by pres-

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[1] *Rosenheim, Deutsche med. Wochenschr.*, 1905, Nr. 27.

[2] *Keith*, quoted from *Lennander*, "Meine Erfahrungen über Appendicitis." *Mittheilungen aus den Grenzgebieten*, 1904, Bd. XIII, p. 326.

sure in the homologous area upon the left side will at once indicate whether we are dealing with a physiologic or an abnormal zone sensitiveness.

An interesting question is involved in the decision whether acute or chronic perityphlitis will in the course of time develop from such latent cases. In so far as I have been able to review the literature, such transitions have not been described, but we must not overlook the fact that these abdominal diseases have been recognized only in the last few years by careful palpation of the cecal region. The analogous occurrence of a latent cholelithiasis or cholecystitis, which I have not infrequently observed, strongly favors a latent perityphlitis. Here I have several times noted the appearance of acute inflammation or typical attacks.

It is highly desirable that clinicians and physicians should devote more attention to the occurrence and the significance of latent perityphlitis than has thus far been the case.

(b) **Typical Cases of Chronic Relapsing Perityphlitis.**—Probably most cases of chronic relapsing perityphlitis set in very insidiously and without prodromes, and gradually the symptoms become more severe. If the patients are asked from what period of time they date their disease but few can give a satisfactory answer, for the disease is of chronic type from the onset.

The symptoms vary greatly in intensity. Sometimes drawing pains in the ileo-cecal region are caused by any motion, or by bending or lifting, or by prolonged sitting; in other cases there are only sensations of pressure and weight in the right abdominal region which are annoying to the patient; sometimes it is merely a sense of fatigue after prolonged walking; sometimes the disturbances are more or less dependent upon defecation, which in such cases is usually irregular; occasionally there are also more or less typical bladder symptoms (tenesmus, painful passage of urine, etc.).

The previously mentioned disturbances now show distinct exacerbations and remissions which are probably to be attributed to the factors of bodily rest and avoidance of exertion of any kind. The occupation of the patient of course here plays an important rôle. Those who are able to take care of themselves sometimes have intervals of fair or even normal health while the symptoms almost invariably increase in those whose circumstances compel them to labor. I have a patient whose case is interesting because his appendicular symptoms were increased after each coitus.

In their *objective* signs, there is a marked difference between residual perityphlitis and the pure, chronic form. This is shown by the fact that in the residual form and during the interval we not infrequently find distinct traces of inflammation which has run its course by palpating exudates, strands, or thickening in the ileo-cecal region. It may be re-

garded as a rule with but few exceptions that when such plastic changes in the region of the appendix can be determined an acute inflammatory process has always preceded.

Only under rare circumstances are these plastic changes found in the true, chronic, relapsing form. Therefore, I do not concur in the views of H. Herz,[1] who has published an otherwise excellent article concerning the condition under discussion and who states that the exudate or the tumor is the unmistakable sign of chronic perityphlitis.

Here I wish to call attention to the fact that the possibility of demonstrating thickening, adhesions, etc., at or around the appendix, is often greatly overestimated. Such adhesive changes may be suspected in the residual processes of an acute perityphlitis. Since they occur with relative frequency they are not rarely met with at a laparotomy, but under such circumstances the diagnosis is merely an accidental one. If honest, we must admit that only a *distinctly palpable tumor* or resistance permits conclusions in regard to residual processes. In my opinion there is rarely a doubt as to whether or not resistance is present, especially in the ileo-cecal region; but there may possibly be a question as to its character (exudate, malignant tumor, ileo-cecal tuberculosis, or induration).

In the overwhelming majority of cases of the chronic relapsing form, we find as an objective expression of perityphlitis the presence of a *painful pressure zone in the region of McBurney's point.*

This can be easily found by drawing upon the body two imaginary lines one of which connects the anterior superior spine of the ilium upon the right side with the umbilicus, and the other outlines the external margin of the right rectus muscle. The point where these lines intersect each other coincides with McBurney's point.

The importance of McBurney's point in the diagnosis of perityphlitis will be more fully discussed in the section devoted to diagnosis. Here I shall only add that the point, or, more correctly, McBurney's zone, is nothing more than a convenient landmark.

There can be no doubt that in chronic perityphlitis McBurney's point is usually sensitive to pressure, and it would be a misrepresentation of facts if we were to ignore this painful zone; in its intimate relation to the other symptoms it is naturally a clinical aid.

When the processes in typical cases of chronic relapsing perityphlitis show acute increase, this is of limited extent. The temperature is but moderately elevated (100.4° to 101.3° F.), and upon the next or the succeeding day falls to the norm. Corresponding with the temperature the pulse remains normal as to quality, it shows no intermissions, and is only moderately rapid. Except for the increase of the ileo-cecal pain the general condition is but slightly disturbed. Signs of peritoneal irri-

[1] *H. Herz, Therap. Monatshefte,* 1905, H. 3 u. 4.

tation are absent (vomiting, meteorism, facies peritonealis, singultus). The ileo-cecal region itself is more sensitive to pressure upon the first or second day of the disease than during the interval, but under suitable treatment this tenderness upon pressure either disappears in from twenty-four to forty-eight hours or ameliorates to the degree present in the intervening period.

(e) **Atypical Cases of Chronic Relapsing Perityphlitis.**—There are numerous variations from the type above described. To enumerate and to describe these would be to include an enormous number of histories. Nevertheless, among the irregular forms there are certain pathologic groups with common and easily recognizable features, and these are the more important in practice because a failure to observe them leads to erroneous therapeutic measures.

Appendicular Colic.—Among the atypical cases colica appendicularis (colique appendiculaire, Talamon) must primarily be mentioned. Under this name Talamon was the first to describe in a publication,[1] which is even to-day a masterpiece, a form of chronic perityphlitis distinguished by the following characteristic symptom-complex: The patients, frequently without a recognizable cause, or occasionally, as they report, in consequence of an error in diet, are attacked by severe colic which extends over the *entire abdomen* (not, as is stated in some text-books, limited to the ileo-cecal region). This is extraordinarily severe and lasts from 6 to 8 hours or longer. During the attack the entire abdomen is more or less sensitive and tense, just as in the case of flatulent colic, but after the attack has passed a sensitiveness in the ileo-cecal region appears and lasts for several days—and this is the most important feature in the entire symptom-complex. This subjective tenderness which becomes noticeable on active movements and even in walking and bending over corresponds to the extreme tenderness in the region of the appendix. But the subjective sensations and the objective sign of tenderness on pressure disappear after a few days, until a renewed attack produces the same symptoms. Fever and other changes referable to inflammation are absent, as a rule, in the ordinary form of colica vermicularis.

What is the cause of these peculiar attacks? Most authors quite properly attribute them to spasmodic contractions of the appendix which result from the attempt of the organ to expel foreign bodies, fecal concrements, or other substances which have found their way into the appendix from the cecum.

A very interesting case of this kind from Wölfler's Clinic was described by Goldbach.[2]

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1 *Talamon*, "Appendicite et Perityphlite." *Bibliothèque médicale, Charcot-Débove*, Paris, 1892.

2 *Goldbach, Prager med. Wochenschr.*, 1898, Nr. 16.

A student, aged 16, had for a year following jaundice severe colicky pains under the right arch of the ribs. During the attack of pain there was never vomiting or fever, but obstinate constipation was present. Later the pains reappeared, particularly in the evening; there was no pain in the morning. At the time of examination the pains were felt in the ileo-cecal region, the pain being definitely localized in McBurney's point. Calculi were never found in the feces. Palpation revealed an oval, somewhat soft tumor (cecum), upon which an elongated, cylindrical body was distinctly palpated. This, as a whole, appeared to be movable, and was sometimes found in the hepatic region, at other times in the lower abdominal region. The liver was not enlarged. At the operation two small stones were found in the cecum. If the stones were forced toward the vermiform process they readily slipped into this organ and just as readily were returned to the cecum, which was absolutely normal. Extirpation of the appendix. Recovery.

Among the atypical cases, MASKED (Treves) and LARVAL PERITYPHLITIS (Ewald) must be enumerated.

Under the name of "*masked perityphlitis*" Treves[1] has described a number of cases in which there were absolutely no signs of perityphlitis, and in which the ileo-cecal region was free from pain. In one of these cases the symptoms were chiefly those of pylephlebitis, while the necropsy showed small hepatic abscesses and (as a primary infection) a completely transformed appendix filled with pus. I recently observed a case, in many respects similar to this, which caused great perplexity in diagnosis.

Mr. O. F., a merchant, aged 22, from Antwerp; his parents were healthy. As a child the patient had measles, diphtheria, and whooping-cough, and five years ago enteric fever. From childhood he had a tendency to diarrhea in consequence of dietetic errors. In June, 1903, while in New Orleans and after a slight alcoholic excess, the patient had a mild attack of fever accompanied by pain in the gastric region and the back and also diarrhea; the attack lasted for a few days and then yielded. In the beginning of August, 1903, a "mild attack of colic" with pains in the abdomen and diarrhea occurred very suddenly (without known cause). In the following months he frequently had diarrhea without pain.

In March, 1904, the patient had an attack of *jaundice*. Prior to the outbreak there was slight malaise for a few days and some fever ("like an influenza"). Pain was not present. After the jaundice had existed for three weeks, pains were felt in the anus and also periodic pains in the genitalia which radiated to the anus. These attacks always lasted from fifteen to thirty minutes. During this time the patient could void his urine only in drops. After a few days the pain passed away, and micturition was normal. The jaundice also gradually disappeared, and in the spring of 1904 the patient completely recovered at Baden-Baden. In July, 1904, diarrhea and vomiting appeared suddenly in the night; at first without pain, but toward morning pains were felt in the entire abdomen, passing away in a few hours. The patient is said to have had no fever. Three weeks later there was sudden severe pain in the "gastric region" (left side of the abdomen), which later radiated to the *right lower side of the belly*; there was mild fever. The physician suspected perityphlitis. After three days the patient was able to resume his occupation. In August there was diarrhea with the passage of mucus and tenesmus. Early in September, 1904, he was again suddenly attacked by chills and fever with severe pains

[1] *Treves*, "Perityphlitis and Its Varieties," London, 1897, p. 37.

in the gastric region (epigastrium) which radiated to the lower abdominal region, also vomiting. An injection of morphin soon relieved the pain, but for a few days there was marked sensitiveness below the right arch of the ribs which to a slight extent had existed since the attack of jaundice in the spring. Since the last attack there has been a tendency to diarrhea alternating with constipation, occasionally mucus in the dejecta, and sometimes mild pain in the right abdominal region (between the hip and the right border of the ribs).

The patient, who had made a journey from Antwerp to Berlin without difficulty, was suddenly attacked the first night with severe pain in the abdomen (in the middle of the belly somewhat above the navel) which soon radiated to the right lower side and the region of the bladder, this being accompanied by bilious vomiting and, toward morning, by chills. The temperature rose to 101.7° F., the pulse was 104, but regular, full, and of good tension. The abdomen was retracted; the walls showed only moderate tension. Just above the navel the belly was very sensitive to pressure. The ileo-cecal region and the other parts of the abdomen were moderately tender to ordinary pressure.

The liver and gall-bladder were not palpable, nor painful on pressure. The splenic dulness was normal, the organ could not be palpated. The urine was clear and free from albumin and sugar. Digital rectal examination was very painful, the prostate gland slightly enlarged; otherwise nothing abnormal could be palpated. Lungs and heart normal.

The fever continued for several days; the pulse was always regular and full, the greatest rapidity being 108. After the 2nd of November the abdomen was moderately distended. Above the navel and also in the region of the sigmoid flexure there was extreme tenderness both spontaneously and on pressure, but pressure in the ileo-cecal region evoked no actual pain, nor was there pain upon pressure in the region of the liver and gall-bladder. The pains above the navel radiated downward to the bladder region. Up to the 4th of November neither feces nor flatus were passed. After intestinal irrigation there was a slight fecal evacuation accompanied by vomiting. Pain was everywhere produced on pressure, and was most marked above the navel. Upon the 5th of November the bowels moved freely; the abdomen was no longer distended but soft, although painful upon pressure above the navel, and somewhat less so in the region of the sigmoid flexure; other areas not painful. The temperature returned to the norm and remained so for a few weeks except for a transitory rise in consequence of catarrhal tonsillitis. The patient constantly had fleeting pain in the abdomen which radiated from the middle of the epigastrium down to the bladder and the ileo-cecal region. Upon pressure in the latter area there was but little pain, but this pressure invariably produced pain in the epigastrium and sometimes in the region of the bladder. At the end of urination also there was frequently slight pain in the bladder. The urine was somewhat turbid: the sediment contained sodium urate and isolated bladder epithelia. The bowels moved daily, the dejecta were formed, and on rare occasions were admixed with a little mucus (immediately after the attack the mucus was considerable). The patient recovered within the next week, gained 5 kilograms in weight, was out of bed, and had taken two carriage drives when, upon the morning of the 12th of December, there was sudden severe pain in the bladder region and in the radix penis accompanied by bilious vomiting. The temperature rose above 100.4° F. The abdomen was retracted, tense, and everywhere painful on pressure, particularly in the bladder region over the symphysis. Pressure over the liver and gall-bladder was not especially painful. At the termination of urination there was intense pain in the bladder which radiated to the epigastrium. The urine showed nothing abnormal. The pulse was 124, and full and strong. Rectal examination and palpation revealed nothing abnormal. After an injection of morphin the pains ceased for a short time.

The temperature continued high for the next few days, the abdomen tense and painful, especially in the bladder region, more so upon the right side than upon the left, and very tender upon pressure. Nothing abnormal could be palpated. The pulse was invariably full and strong, the pulse rate as high as 132. Up to December 15th, notwithstanding oil enemata and the introduction of an intestinal tube, neither feces nor flatus were passed, and upon the 14th vomiting and eructations were frequent. Upon the 15th of December, after two profuse intestinal irrigations with soap-suds a large amount of feces and flatus was discharged. The abdomen became soft. The bladder region was still painful upon pressure. The temperature fell to normal. Sensitiveness in the epigastrium (spontaneous and on pressure) continued, as well as severe pain on pressure immediately over the symphysis, and more marked upon the right than upon the left side. The ileo-cecal region and the region of the liver and gall-bladder showed no tenderness. The abdomen revealed nothing abnormal upon palpation or rectal examination. At the termination of micturition, as well as prior to defecation, there was still occasional pain in the bladder region. *A tentative diagnosis was made of appendicitis, the appendix being displaced downward and adherent to the bladder.* Operation was advised. Upon the 25th of December there was another slight rise in temperature. On the 27th the patient was admitted to Prof. Körte's Clinic, where, in an examination under an anesthetic, nothing abnormal could be found. Operation was performed by Prof. Körte upon the 2nd of January, 1905. Under chloroform anesthesia an incision was made in the median line above the navel and extending two fingerbreadths above the symphysis. The omentum was found to be adherent to the pelvis, the cecum adherent to the bladder, the appendix was displaced downward into the pelvis behind the bladder and there adherent to the bladder. The adhesions of the omentum to the bladder were broken up as well as the adhesion of the omentum to the sigmoid flexure. Upon loosening the appendix, perforation occurred at the base of the organ and a fecal calculus exuded. Amputation of the appendix. In the pelvis a small abscess was opened in loosening the adhesions. Coils of the small intestine adhered to each other, and their dissolution was impossible on account of the extent of the adhesions. The serosa of the intestines was reddened; in some areas there were small whitish nodules (tubercles?). Gall-bladder: No adhesions; no stones could be palpated. In the liver, so far as it could be palpated, there was nothing abnormal. The abdominal walls were sutured with drainage. Near the base of the appendix which was opened an ulceration the size of a pea was found, and this had perforated when the organ was removed. The appendix contained purulent feces. The muscularis and submucosa were thickened.

Recovery was slow, but there was no complication except a suture abscess. The highest temperature range was from 98.6° to 100.4° F. Upon February 18th, 1905, the patient was discharged well. The intestinal function was somewhat torpid but otherwise normal.

According to Treves, in numerous other cases there are no symptoms which point to perityphlitis, but they chiefly indicate general dyspepsia (loss of appetite, constipation, occasional vomiting). The abdomen is somewhat distended, feels tense, and is frequently the seat of indefinite colicky pains. Fever is either absent or is but slight. Upon deep pressure in the right iliac fossa there may possibly be some sensitiveness. The swelling may be in the impacted cecum, the intestinal contours being sharply defined. The cases run an indefinite course until the diagnosis is confirmed by the detection of an abscess.

As is obvious, the group last described bears a certain similarity to the cases designated by Ewald [1] as PERITYPHLITIS LARVATA. In this form also there are atypical gastric and intestinal symptoms of varying nature and intensity but, in contradistinction to the masked cases of Treves, in perityphlitis larvata there is an evident sensitiveness to pressure in the ileo-cecal region. Such atypical cases are by no means rare, and can be correctly diagnosticated only when, as already remarked, the area of the appendix is carefully palpated in every case of visceral disease.

In my article quoted above [2] I have called attention to another variety of atypical perityphlitis distinguished by this feature: That the patients complain little of distress in the ileo-cecal region but, on the contrary, refer their symptoms to the hepatic region, while here objective examination reveals normal conditions, but extreme sensitiveness to pressure at McBurney's point.

COMPLICATIONS OF CHRONIC PERITYPHLITIS

In the residual as well as in the relapsing forms of perityphlitis complications frequently set in. In the former they may under some circumstances so completely dominate the pathologic picture that the actual focus of the disease is masked. To this category belong the dreaded hepatic abscess and pyopneumothorax subphrenicus, secondary pleurisy and pneumonia, and the embolic processes in the lungs which develop in the course of, or after an operation for, acute or residual perityphlitis.

This by no means exhausts the number or the varieties of the complications; were we to attempt at this point to give a comprehensive description of this grave disease which so frequently threatens life, it would be necessary to describe the entire pathology of acute perityphlitis.

We shall limit ourselves to mentioning a restricted number of the complications of chronic perityphlitis.

Here two groups are alike interesting: Chronic catarrh of the colon and (in women) diseases of the right-sided adnexa.

In regard to the first, catarrh of the colon in its various forms and manifestations is among the most frequent and, we may almost say, daily accompanying symptoms. Here we must discuss the question in how far colitis is the cause or the effect of chronic perityphlitis. We must admit that catarrh of the colon precedes as well as follows perityphlitis, and in such a manner that the bland diet necessitated and the lack of sufficient bodily exercise leads to habitual constipation and gradually to true catarrh.

The second group of complications relates to diseases of the female genital organs, especially of the right-sided adnexa. It is not within my

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[1] *Ewald*, "Die Krankheiten des Darmes und des Bauchfells." Berlin, 1902, p. 237.

[2] *Boas, Deutsche med. Wochenschr.*, 1905, Nr. 27.

province to describe minutely these serious affections. Those who desire detailed information are referred to the instructive and exhaustive monograph of Th. Landau, which is a critical analysis of extraordinary merit.

According to Th. Landau, besides the true diseases of the adnexa, the ovarialgia, the retroflexioversio uteri mobilis, and the dislocated right kidney may be combined with perityphlitis, and thus mask the actual underlying affection.

Besides these most common maladies, there are many others which may accompany perityphlitis. First to be mentioned here are calcareous processes of the right kidney and of the gall-bladder, which, more frequently than we are prone to believe, cause errors in diagnosis. Furthermore, as already stated, there may be a dislocated right kidney as well as perityphlitis, and if the symptom-complex be not typical this may make a differential diagnosis either perplexing or impossible. We shall discuss these difficulties more minutely in the section devoted to diagnosis.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF CHRONIC PERITYPHLITIS

The diagnosis of chronic perityphlitis may be easy, even to the novice, or, inversely, may present to the most experienced great and occasionally even insurmountable difficulty.

Starting from the principle, which has been reiterated, that residual perityphlitis and chronic relapsing perityphlitis are two clinically different forms, we shall enunciate the fundamental laws regarding the first mentioned group.

Here, primarily, the history must be regarded as the decisive factor. The more clearly this reveals the occurrence of a prior typical attack the more readily can we decide as to the nature of the possible acute relapse. Nevertheless, experience shows that milder attacks are frequently forgotten by the patient, especially if they have occurred a long time previously. If the patient is a very young person, we frequently find that such mild attacks have not been recognized by the physician owing to the insignificant symptoms, hence in a given case an existing attack may not be regarded as a relapse but as a primary attack.

Aside from this, in all essential points the diagnosis of residual perityphlitis in its acute form is evolved as in the primary attack. Besides this, however, as mentioned before, the chronic residua must be considered. In the discrimination of these the prior history of the patient is the best guide. The occurrence of fever and of the febrile movement, the duration and severity of the attack, and possible complications are especially valuable aids to the recognition not only of the nature but also of the intensity of the process.

These are the cases also in which, following the acute attack, objective symptoms persist which can be demonstrated with more or less accuracy.

The region of McBurney's point is probably sensitive to pressure, but in its surroundings we find at the same time a certain amount of thickening which may be demonstrated even on very slight percussion. In some cases an exudate may be felt. According to Ewald [1] and Sonnenburg [2] this is said to become prominent after inflation of the colon.

Sometimes the differentiation between an exudate and a neoplasm is extremely difficult, and casuistry furnishes a number of instances in which laparotomy or the necropsy has revealed numerous fundamental errors and confusions.

Among the most serious conditions leading to mistakes are carcinoma of the cecum and ileo-cecal tuberculosis.

In fact these affections, provided positive symptoms and a clear history do not guide the physician, run their course with manifestations which closely resemble chronic perityphlitis.

In the following we will present a few principles for the differentiation of these clinical pictures the prognosis of which varies so greatly, but in which, however, we cannot deny that no differentiating factors will invariably protect us from error.

Cecal Tuberculosis.—Cecal tubereulosis usually occurs in comparative youth (from the second to the fourth decade), runs an exceedingly chronic course, and frequently, although by no means always, is accompanied by tuberculous processes in the lungs. As Obrastzow correctly points out, the cecum does not form an actual tumor, but has the appearance of a rigid, irregular, nodular infiltration. Cecal tuberculosis invariably runs its course with symptoms of stenosis which in such cases are revealed in a typical manner (well developed intestinal peristalsis, alternation between constipation and diarrhea, attacks of obstruction). Blood and pus are rarely present in the feces, but tubercle bacilli may be frequently found. Fever is rare.

Carcinoma of the Cecum.—As a rule, in careinoma of the cecum which usually occurs late in life the tumor is sharply limited. Symptoms of stenosis may be absent, or may be present in a greater or less degree. Fresh or occult blood (Boas [3]) appears in the feces in from 10 to 15 per cent. of all cases; pus is either not found or is exceedingly rare. Fever may occur in carcinoma of the cecum, but only in advanced stages of the affection.

Where there is no thickening, tumor formation, or even symptom of stenosis, the only objective sign is sensitiveness to pressure in the ileo-cecal region.

In rare cases, to find at the autopsy an inflammatory or even malignant

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1 *Ewald*, "Die Krankheiten des Darmes und des Bauchfells," Berlin, 1902.

2 *Sonnenburg*, "Pathologie und Therapie der Perityphlitis," 5 Aufl., Leipzig, 1905.

3 *Boas*, "Ueber occulte Magen- und Darmblutungen." *Volkmann's Sammlung klin. Vorträge*, 1905, Nr. 387.

tumor instead of the expected perityphlitis is an experience from which even the most skilful diagnostician is not exempt.

In these and other doubtful cases I must call attention to a factor that aids materially in the diagnosis of residual perityphlitis: This is a regular temperature record continued for weeks, and especially the comparison of temperatures taken simultaneously in the axilla and in the rectum. A copy is here presented of a temperature curve which clearly portrays these differences.

I have been several times convinced that, under some circumstances, these more or less high elevations of temperature, and especially the simultaneous rectal and axillary temperatures, form the only guiding and determining symptom. The following case may serve as an example:

A girl, aged 4, daughter of a manufacturer from G. Following a doubtful acute perityphlitis she complained of periodic abdominal pains which lasted but a few seconds and occasionally ceased after vomiting. Localization of the pain was impossible. The objective finding was entirely negative, the region of the cecum being painless upon pressure. Regular and continuous records of the temperature showed occasional falls or elevations of brief duration. A diagnosis of residual perityphlitis was accordingly made and the child was operated upon by Prof. Körte. The operation revealed the following: In the mesentery a swollen gland which was extirpated. The appendix was about 10 cm. in length, contained semisolid feces, and in the center a mucous membrane cicatrix. The cecum and ascending colon were palpated and found to be normal. Course normal; discharged cured.

While in residual perityphlitis the prior history will usually give us valuable data for the diagnosis, in the *chronic relapsing form* we must depend principally upon the clinical course. Frequently this ensures a diagnosis; but there are also cases so faintly characterized that only repeated and thorough examinations will enable us to come to a decision. Among the diagnostic factors which favor chronic perityphlitis the most prominent are the characteristic subjective symptoms: The *persistent* pains starting from the ileo-cecal region and centrifugally radiating to the thigh, to the lumbar and the hepatic regions. Among the objective symptoms, as already mentioned, there is frequently nothing but the sensitiveness at McBurney's point.

In some cases another phenomenon appears upon palpation, which at this point must be described somewhat more explicitly since it is frequently and variously referred to in literature: This is palpation of the diseased vermiform process. The American surgeon, Edebohls, deserves credit for having called attention to the possibility of palpating the diseased vermiform process, and in 1894 he made the statement that this organ could be recognized in every case of perityphlitis.

That this assumption is not borne out by the facts can to-day scarcely be doubted by physicians of experience. In cases of acute perityphlitis the swelling of the mesentery, the appearance of the exudate, etc., make the conditions for palpating the diseased appendix extremely unpropitious.

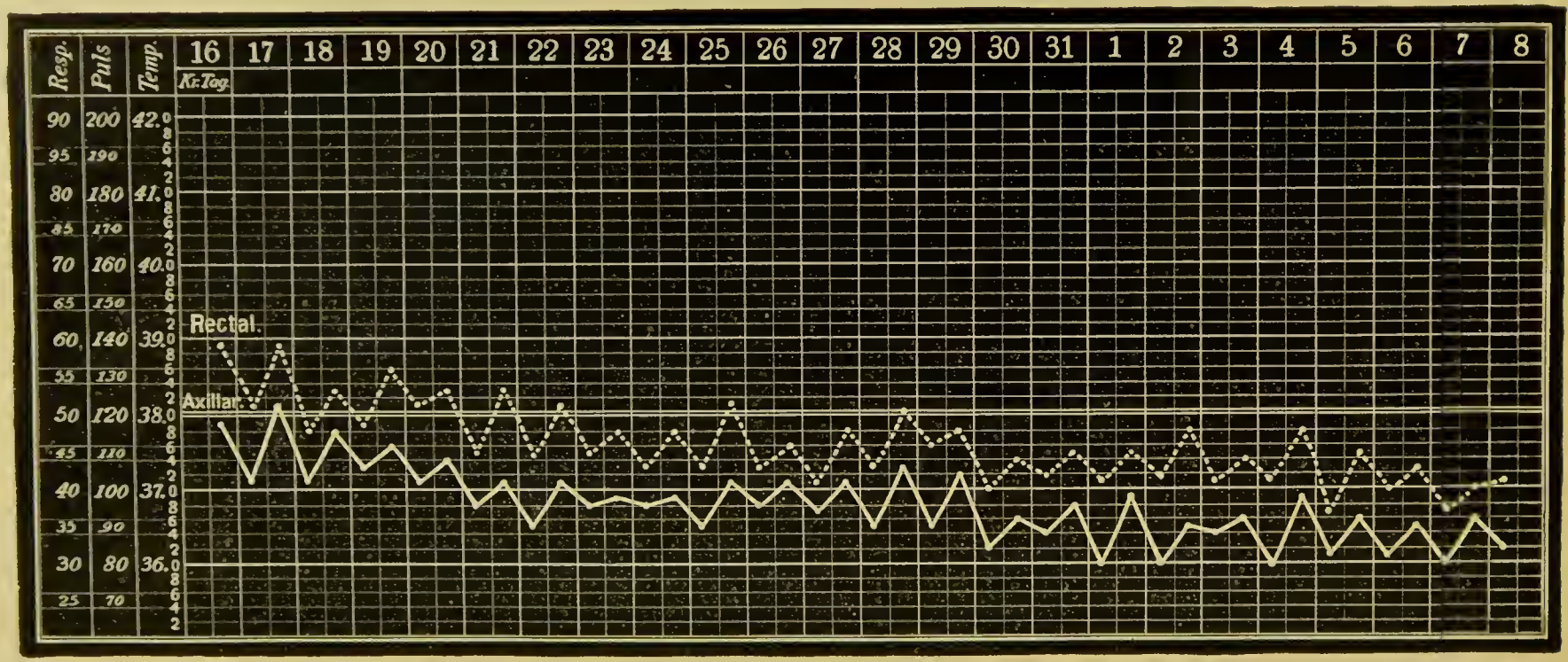

Fig. 21.—Diagnosis: Perityphlitic Exudate (relapse).

Axillary temperature: ———— Rectal temperature: .....

The circumstances are somewhat more favorable for palpating the appendix in chronic perityphlitis, a fact to which some authors (Ewald, Hausmann) have attached a certain diagnostic significance.

In these instances we may admit that now and then when the vermiform process is in a favorable position it may be felt as a round, painful organ, sometimes with surprising distinctness. Nevertheless, dozens of cases of chronic perityphlitis may be observed in which the result of palpation is not only once but repeatedly negative. In other cases in which we think that something is palpated, the body that is felt cannot be clearly recognized, and we waver in opinion between the assumption of a contracted portion of the colon, of a coil of the ileum, of adhesive intestines, or of the rolled-up mesentery, etc.

At all events, in my experience palpation of the diseased appendix is only of diagnostic importance if we constantly find in the ileo-cecal region a distinctly palpable body which is sensitive to pressure, and which is of the same form and thickness as the vermiform process. In such isolated cases, of course, the diagnosis may be much more readily made.

The actual difficulties in the diagnosis of chronic perityphlitis depend upon our distinguishing this from other chronic forms of colitis, as well as upon the differentiation from chronic disease of the uterine adnexa.

In the great majority of cases the condition in the former is such that we must decide whether merely colitis is present or whether this coexists with chronic perityphlitis.

The differentiation is extremely difficult when the chronic colitis has its seat in the cecum, i. e., when a true chronic typhlitis exists. Then a symptom picture may develop which nowise differs from chronic perityphlitis. As a matter of fact the diseases may exist simultaneously.

The condition is somewhat more favorable when the colitis occurs in a more peripheral section of the colon, and when a severe but circumscribed pain upon pressure is produced in the ileo-cecal region. If the clinical picture of perityphlitis is simultaneously present, we can hardly doubt their coexistence.

The diagnosis is facilitated if acute, mild, febrile exacerbations appear similar to those which occur in the course of chronic relapsing perityphlitis; this impresses the stamp of certainty upon the previously doubtful case.

Nevertheless it cannot be denied that in a not inconsiderable percentage of cases doubts will arise which cannot be removed even by careful investigation and observation.

To this category belong, for example, the apparently not rare cases of so-called *pseudo-perityphlitis* to which Nothnagel [1] first called attention, and which all experienced physicians have met with.

[1] *Nothnagel. Wien. klin. Wochenschrift*, 1899, Nr. 15; compare also *G. Singer*, "Pseudoappendicitis und Ileocöcalschmerz," Wien und Leipzig, 1905.

We are not always in a position to verify our diagnosis by operation, as was the case with Nothnagel. Nevertheless, there are some symptoms which may be looked upon as landmarks in the differential diagnosis between true and pseudo-perityphlitis. Paramount among these is the course: At certain irregular intervals the patients suffer from pain in the ileo-cecal region, but this is not associated with rises in temperature and is not increased by active or passive movements. The region of the appendix is either insensitive to pressure or the sensitiveness is only referred to the skin, and it is not circumscribed but diffuse. Occasionally cutaneous hyperesthesia alternates with normal sensation upon pressure.

I recently had an opportunity of observing a very instructive case of well-developed *typhlophobia*. In a family which was well known to me for years a fatal case of severe acute perityphlitis had occurred in a near relative. This case was for weeks the subject of conversation in the family, and one of the two daughters of the house, a girl aged fourteen, became especially concerned and uneasy. A short time afterward she complained of severe pain in the region of the cecum, lost her appetite, remained away from school, became anxious, and caused her mother a great deal of worry. The history showed that the child had never had fever. Examination of all parts of the abdomen revealed absolutely no sensitiveness nor resistance, and the adduction of the thigh which was several times attempted produced no pain. This proved that there could be no question of perityphlitis. I quieted the anxious patient, and advised her to return to school the next day and to consider herself as well. From this moment the child was well and remained so.

Besides organic and neuralgic intestinal affections, in the female the differentiation of diseases of the adnexa may come into question in a diagnostic respect. We have already indicated the possibilities which exist, and at this point attention must be called to the necessity of considering these symptomatically with chronic perityphlitis, for these genital affections may be quite analogous. In the diagnosis of inflammation of the adnexa a considerable degree of practice and experience is a prerequisite, therefore in cases at all doubtful the opinion of a gynecologist should be sought.

Besides these factors which dominate the differential diagnosis of chronic perityphlitis, there are still others which now and then come into question.

Among these are cases of calculus in the right kidney or of the *uric acid diathesis*. The regular finding of abnormal amounts of uric acid combined with small amounts of albumin in the urine, the appearance of typical attacks with their characteristic urinary changes, the demonstration of a gouty substratum in characteristic areas of the body, and the palpation of the kidney itself will in many cases reveal the correct diagnosis. Nevertheless, the *ensemble* of the previously mentioned symptoms is

not always so favorable that the underlying condition can be clearly discerned.

With reference to the kidney, its dislocation, at least so far as subjective factors are concerned, may produce symptoms which to a certain extent resemble those of chronic perityphlitis. But no actual attacks are observed, there is no typical pressure point in the ileo-cecal region, and the characteristic localization of the pains is also lacking.

Atypical cases of cholelithiasis and of cholecystitis may be confounded with chronic perityphlitis, particularly as they are not infrequently combined in the same individual. To this must be added another source of error, the not rare coexistence of cholelithiasis and colitis. An accurate history combined with cautious and repeated examinations of the liver and the region of the gall-bladder and the occasional finding of biliary coloring matter in the urine will often clear the complicated picture.

Finally, we must mention the not infrequent possibility of error from the presence of ill-defined hernia, particularly of right-sided inguinal and crural hernia. I have several times seen cases of supposedly chronic perityphlitis which were permanently cured by wearing a hernial truss.

This by no means exhausts the number of perplexities which may arise in differential diagnosis, but from what has been stated it is obvious that in some cases the diagnosis of chronic perityphlitis requires considerable acumen, practice, and experience.

One factor in particular must not be omitted from consideration, namely, that chronic perityphlitis may appear simultaneously with or independently of diseases of other organs. Sometimes we are inclined to follow an old rule and to substitute one or more pathologic varieties for each other. But nature does not sanction such a method, and we must therefore consider not only whether one or the other affection is present but—this is often the most difficult problem—whether they do not exist independently of each other.

PROGNOSIS OF CHRONIC PERITYPHLITIS

In this article we have repeatedly called attention to the fact that the prognosis in residual and in chronic perityphlitis presents fundamental differences. This is evident from the etiology of both forms of the disease. While, as is shown by the fitting designation of Sahli, in acute perityphlitis there is always "a purulent nucleus," the changes in the chronic form are referred more to the mechanical realm, and consist of kinking, thickening, adhesions and obliterations.

This is the reason why the process in acute perityphlitis may heal functionally, for example, by self-drainage (Sahli), or by rupture into

the intestine or into other organs. The conditions are, however, different in the cases with a chronic beginning and running a chronic course. Here mechanical disturbances when once developed to such a height as to produce symptoms rarely or never heal. From the fact that even when acute perityphlitis terminates in recovery it leaves residua of varying extent and absolutely unlike quality, it follows that the prognosis of residual perityphlitis precludes any *a priori* judgment. Experience also teaches us that the primary attack may yield without any difficulties and complications while the second attack may develop in an extremely fulminant manner, and if timely surgical aid is not at hand may cost the life of the patient. And, *vice versa,* the first attack may be exceedingly dangerous while the following ones may run a smooth, mild course.

In such cases, any attempt to lay down definite rules and to institute a plan of treatment in accordance with these, in opposition to the expressive voice of practical experience, must be unwise and even dangerous. Here and there, it appears as if certain laws in the course of residual perityphlitis were decisive, but this is nothing more than an accidentally favorable sequence in the individual cases, and with some patients it collapses like a house built of cards.

The prognosis of chronic relapsing perityphlitis is much more certain than that of the residual form. With few exceptions the prognosis of the latter, in so far as life is concerned, may be regarded as favorable. This is true of the intervals as well as of the attacks which occur periodically. Although I am well aware that no conclusive significance attaches to a single observation, nevertheless the following clinical history may be here in place because it shows how even extensive and chronic disease of the vermiform process may set in yet not decidedly impair the organ.

The case was that of a lawyer, aged 25, who in the year 1904 began his voluntary service as a soldier in the army. In 1905 he presented himself to me for treatment showing all the signs of chronic relapsing perityphlitis. Besides this there was an obstinate habitual constipation with colitis. Although I warned the patient of the incompatibility of military duty with his disease, he insisted upon continuing his service in the army. He passed energetically through the difficult period of his recruit service; gradually, however, his symptoms increased so that in April, 1905, I strenuously advised operative removal of the appendix. Even then his condition was still bearable, and only the fear that in all probability he could not serve his time induced him to consent to the operation, which was successfully performed by Prof. Körte upon the 21st of May, 1905. The appendix was long, its end thickened, and it contained fecal concrements. Microscopic finding: Appendicitis ulcerosa.

Notwithstanding the strenuous corporeal exertion to which the patient was exposed during his military career, and although he punctiliously performed all of his duties, no true attack supervened, which is certainly a forcible illustration of what has been said above that chronic relapsing cases show no tendency to a severe course.

The prognosis in regard to a cure is quite different. While in an acute attack, even when most severe, a cure in the ordinary sense ensues and may be permanent, in the chronic relapsing form, for reasons previously mentioned, the result is quite different. Since in this form the symptoms at the onset are frequently very slight, it cannot be denied that by immediate and suitable remedies which will be described in the chapter upon treatment the disturbances may be so lessened as to constitute a cure; but a true cure which under observation lasts for years I am not cognizant of,[1] and in such a case I should be inclined to doubt the correctness of the diagnosis.

The most characteristic feature of chronic perityphlitis, not only in its course but also in its prognosis, is the fact that under favorable external conditions (bodily rest, lessening of tension, mineral spring cures, etc.), distinct remissions occur. But this somewhat subjective euphoria is in sharp contrast with the objective investigation which shows that a distinctly recognizable sensitiveness to pressure is constantly present in the ileo-cecal region.

TREATMENT OF CHRONIC PERITYPHLITIS

In the borderland between internal and operative treatment, the treatment of chronic perityphlitis most frequently lies. *It is everywhere recognized that the most radical and relatively the least dangerous method of cure of chronic perityphlitis consists in the extirpation of the appendix in the afebrile stage.*

Upon superficial observation any further discussion as to the best mode of treatment of chronic perityphlitis would appear to be superfluous. But upon accurate weighing of the conditions they are found to be not quite so simple, and it is therefore necessary most carefully to investigate this question.

In the first place, in the therapeutic indications and contra-indications there is a fundamental difference between those of residual and chronic perityphlitis.

As we have seen, an acute attack may result either in complete recovery (in a functional sense) or the residua may generate chronic symptoms, or, finally, may lead to a subsequent attack.

The more resultless the investigation of the anatomical relations the more positively is therapeutic treatment indicated.

We must, therefore, decide to what extent the changes after an attack

[1] *Albu*, in an article recently published, "Zur Diagnose und Therapie der chronischen Perityphlitis" (*Deutsche med. Wochenschr.*, 1905, Nr. 25 u. 26), mentions two apparently cured cases of residual perityphlitis. But in both cases the period of observation was too brief to permit a definite opinion.

are perceptible, and in how far we may prognosticate with probability the type of subsequent relapses.

Considering first the residua, the presence of an exudate may be certainly determined and its growth or retardation demonstrated by continuous observation. On the contrary, exact knowledge of the condition of the vermiform process and its immediate surroundings is much more difficult to acquire. It may still contain pus, a tendency to perforation and ulceration may still be present, the mesenteriolum may be inflamed or gangrenous, yet there may be no marked or grave symptoms to indicate the gravity of the condition. Inversely, a slowly progressive healing may take place, but whether this is permanent is a question to be answered only with the greatest reserve.

As we have already emphasized, the prognosis in a second or third attack is most perplexing. We have only to consult one of the numerous clinico-surgical reports upon appendicitis to be convinced that here every rule finds its paradigm in the inexhaustible variations of the clinical course of perityphlitis. Only ripe experience, as has often been demonstrated, warrants the following prognosis: The longer the interval after the first attack the less the danger of subsequent and severe relapses. But even here, as every one who has followed the literature of perityphlitis knows, exceptions now and then occur.

To find the proper indication for our therapeutic procedure, we must consider impartially what internal medicine has accomplished in the treatment of residual perityphlitis. There can be no doubt that the exudative form is a relatively favorable one for treatment. In such cases, indeed, local compresses of peat or salt and peat and salt baths have actually caused the exudate gradually to disappear, and thus the symptoms were either entirely relieved or diminished to a great extent.

Where there is no exudate, the result of this or any similar form of treatment is much less certain. We must be careful not to mistake a periodic amelioration of the process for recovery. As a rule, however, there is nothing to cause us to regard too optimistically an improvement in chronic perityphlitis—this is true of both forms. We have no criterion for deciding whether a process in the ileo-cecal region has healed or whether it is only latent.

For the prevention of new attacks a number of methods have been suggested. These consist mainly of bodily rest, the careful regulation of the diet, the prevention of an accumulation of feces and gas in the intestines, the long-continued use of compresses of various kinds and of different temperatures, etc. For obvious reasons it is very difficult to decide whether these measures are actually of any use in preventing relapses. Unquestionably the most important of these factors are bodily rest and proper care.

But when we regard these and similar prophylactic measures in their

proper light, in the majority of cases we will cling to no illusions concerning them, even if it were possible and practicable to carry them out energetically, whereas in most cases they cannot be employed at all.

It is evident from this discussion that the operative treatment of residual perityphlitis is from principle to be preferred to any other mode of relief. Only in those cases in which we find an exudate and in which the other clinical symptoms appear favorable may we temporize. The same is true of the residua, which may exist for many years and produce no important symptoms.

After recovery from a severe attack we are placed in a position of great responsibility.

To temporize here means to allow the patient to run risks which prognostically cannot be estimated. On this point I am much more in favor of surgery than many surgeons, and I believe the future will show that I am right. Even with attacks of medium severity, I advise an operation in the interval. An expectant treatment should be instituted only in those mild cases which show but slight elevation of temperature and which terminate in recovery in a few days. Of course the individual circumstances of the patient must be most carefully considered, especially his occupation, since prolonged bodily rest is an absolute necessity.

A decidedly more favorable perspective is afforded by the chronic relapsing cases. If we bear in mind the previously stated fact that the overwhelming majority of cases of perityphlitis running a chronic course assume no life threatening aspect, we are justified in employing the remedies of internal and expectant treatment to the fullest extent.

Here also bodily rest and careful nursing are the first considerations. How long rest should be insisted upon depends, in the first place, upon the intensity of the process, and, secondly, upon the extent of time which is possible in the individual case. In the latter respect, as we have stated, our advice is disregarded more frequently than is desirable. In such cases rest for at least a part of the day or upon holidays may be insisted upon. Distinct remissions are frequently observed after several weeks of absolute rest in bed; in other cases, however, these either do not occur or are only of brief duration. And here the factor previously mentioned, the special regulation of the entire digestive function, comes into consideration.

In acute attacks there can be no doubt that indigestion plays a decidedly causative rôle; chronic indigestion also increases chronic inflammation, and *vice versa*. At this point, therefore, a few practical suggestions for the treatment of intestinal disturbances are not out of place.[1]

Primarily, as everywhere else in gastrointestinal pathology, the diet must be so regulated as to be adapted to the character of the intestinal affection. In simple, habitual constipation we institute a diet which will

[1] See also *Boas*, "Constipation and Hemorrhoids," in this volume.

actively stimulate intestinal peristalsis, and one in which sugar and very sweet substances are prominent. The same result is accomplished by food containing much fat; only such articles, however, should be employed which at a low melting point are to a great extent free from fatty acids (olive oil, butter, cream, almond milk), and these constitute important aids. Organic acids, used with discretion, are of great use (buttermilk, sour milk, and the juice of apples, oranges, and lemons).

It is much more difficult, however, in cases of chronic perityphlitis to decide the question whether food which leaves much residue and raw fruits are to be allowed or prohibited. Traditionally we are inclined to prohibit these strictly. Whether this is necessary to such an extent as is nowadays generally maintained is doubtful if we consider that an actual residue of food in the appendix is an extremely rare occurrence. In all cases the prohibition of raw fruit should extend to only those varieties which contain small seeds; vegetables, particularly those which cause fermentation, should be absolutely avoided or given only when suitably prepared. No decided objection can be raised to the use of rye bread, provided it be well baked.

The prohibition of highly carbonated drinks, of the immoderate drinking of cold fluids, and, finally, of the use of ice cream and fruit ices is quite justifiable. Here the interdiction of strong acids (vinegar), as well as of mustard, horse-radish, etc., should also be mentioned.

In case the previously mentioned diet does not regulate the bowels the most effective and least irritating treatment is by oil enemata. On principle I am opposed to actual purgatives, and only under exceptional circumstances do I advise temporarily even the mildest laxatives, such as castor oil, rhubarb, flowers of sulphur, the preparations of sagrada, and Carlsbad salt. Powerful purgatives or those which are of uncertain action are absolutely prohibited.

The treatment of habitual constipation combined with chronic intestinal catarrh is based on the same principles as that of colitis membranacea. On the other hand, the cases of catarrh of the colon running their course with habitual diarrhea must be treated by a régime the opposite of that mentioned above. Beyond a certain extent sugar and milk, perhaps eggs also, must be entirely prohibited, while, on the other hand, meat, fish, mucilaginous soups (rice, sago, barley), red wine and elderberry wine, and acorn cocoa are recommended. Fats of good quality are permissible. Here also carbonated drinks as well as all kinds of beer, ice cream and fruit ices must be avoided. In simple colitis these measures will usually be sufficient; but, if not, the well known astringents and, secondly, intestinal irrigation with astringent solutions must be considered.

Besides rest and a regulated diet, mineral spring and bath cures come into question; although they are often only palliative and transitory, there can be no doubt that these sometimes produce favorable results because

individual curative factors are readily combined in this treatment, which is difficult to attain in treatment at home.

In the rare cases in which the intestinal canal is absolutely intact, we may limit ourselves to baths, salt baths, or peat baths; in others it is better to combine these with spa treatment. For the latter the waters of Carlsbad, Marienbad, Homburg, Kissingen and Wiesbaden are especially suitable. By drinking the spring water at home, or by baths, the less well-to-do classes may attempt to substitute for these cures. After the cure it is well to continue the local peat and salt compresses for a long time. The same effects may be produced by hot linseed or oatmeal poultices, as well as by compresses of absolute alcohol, after which a more or less distinct remission in the inflammatory symptoms will be observed.

Although it is undoubtedly true that these cures energetically and persistently carried out exert a favorable and palliative effect, yet the ultimate curative result is uncertain. Now and then long-continued improvement takes place and resembles a cure, but in the overwhelming majority of instances we merely attain a certain condition of latency. For some individuals this is very desirable, particularly for those who from the onset show less serious disturbances or who are in comfortable circumstances. For the remainder, however, even after the most thorough trial of this or similar cures, the result is merely armed neutrality. After long or short intervals of rest, disturbances appear, at first mild and of brief duration, but gradually becoming severe and continuous.

After considering all of the questions which here arise, we cannot refrain from expressing the opinion that the operative removal of the diseased appendix is, on principle, not only the most radical but also the most effective measure to bring final relief; of course, with certain limitations which must be carefully discussed.

First, I must reiterate that the indication for the operative removal of the appendix in chronic processes is never urgent. We may quietly await the results of thorough palliative treatment. Although, as has been stated, absolute recovery is rare, yet remissions are often very decided. Frequently we find we are dealing with appendiceal neurasthenics who torment themselves and their friends with groundless fears. Unfortunately these fears are at times augmented by some surgeons who grossly exaggerate the dangers of chronic perityphlitis. I have met a large number of these appendiceal hypochondriacs, and it proved no easy task to convince them of the mildness of their affection.

But even with more serious symptoms the indication for operative treatment is not always pressing. This is especially true in the case of aged and debilitated persons in whom any laparotomy is fraught with more or less danger. Here may be enumerated the cases with complications on the part of the heart, the kidneys, the lungs, or with diabetes and the like.

On the other hand, there are cases in which operation is undoubtedly

indicated; for example, in chronic perityphlitis at the age of puberty. Even with relatively slight symptoms it is our duty in such cases to advise operation, as here the bodily rest insisted upon is directly opposed to the no less important bodily development.

The same is also true of those whose symptoms preclude their following their occupations. Here also the intensity of the symptoms must be subordinated to the fact that such persons are permanently unable to earn a living. Naturally the indication is more urgent if the symptoms become extremely aggravated and are but little or not at all ameliorated by palliative measures.

With every indication for operation it is presupposed *that the diagnosis is positive or at least extremely likely.* Errors on account of existing complications, such as intestinal catarrh or disease of the right-sided adnexa, will frequently be unavoidable; nevertheless it is invariably our duty to exhaust all of the diagnostic aids and methods at command before coming to a definite decision.

Before concluding we must discuss the dangers and results as well as the possible sequelæ of an operation. In regard to the first, statistics quite uniformly show a mortality of from one to 2 per cent. Many surgeons, however, perform hundreds of operations without a fatal case. Taken all in all we may therefore designate an operation for uncomplicated chronic perityphlitis as one relatively free from danger.

It is different with *the results.* In the first place, the influence of the operation upon the appendix and upon the remaining intestine which, as already mentioned, is frequently implicated, must be considered separately. While in the great majority of cases the effect upon the appendix is salutary, this cannot be maintained for the remaining intestinal canal. Yet we must reiterate that after removal of the appendix the cure of an intestinal affection is much more rapid because the physician need no longer take into consideration the restrictions which were previously necessary (rest, gymnastics, massage, etc.).

Nevertheless, in a few (but, as we must admit, the minority) of the cases of chronic perityphlitis, increasing experience proves that the actual curative results are by no means satisfactory.

But that I may not be suspected of disparaging the great successes of surgeons in this realm, I must call attention to a recent communication from one of the most distinguished operators for perityphlitis, namely, the English surgeon, Treves.[1]

Among 231 cases operated upon in the stage of latency, in 15 recovery was incomplete. In these 15 cases ventral hernia and all of the disturbances due to healing of the wound are excluded.

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[1] *Treves*, "The Prospects and Vicissitudes of Appendicitis After Operation." *British Medical Journ.*, 1905, March 4.

Among the causes of failure were incomplete removal of the appendix (2 cases), disturbances on the part of the ovaries (9 cases), chronic or relapsing colitis (8 cases), continuance of local pain (7 cases), neurasthenia or hypochondria (5 cases), persistent pain from gall-stones (3 cases), from colic (2 cases), wandering kidney (2 cases), renal calculi (one case), soft masses in the right iliac fossa (5 cases), unknown cause (one case).

In the two cases in which the removal of the appendix was incomplete, this was removed at a second operation.

The recent reports of Haberer[1] from von Eiselberg's Clinic in Vienna are similar. Among 96 cases of operation during the interval, all of whom survived, in only 50 were the symptoms relieved by the operation. *"Forty patients, however, continued to have more or less marked disturbances, in some stubborn constipation, in some severe pain, even attacks of colic, such as they had suffered from prior to the operation."*

Reports of relapses after operation for perityphlitis are increasing in literature.[2] One of the first authors to report these occurrences was Gerhardt.[3]

I, too, have observed 5 cases of post-operative typhlitis, some of which were very obstinate. I shall quote one case, that of a colleague:

Dr. H., of F., aged 38, suffered for many years from constipation. In 1901 he had an acute attack of perityphlitis with fever and subsequently, at intervals of four weeks, five attacks, all running their course with slight fever. He was operated upon in March, 1902 (Professor v. Czerny, Heidelberg).

The conditions found were as follows: The appendix was bent, twisted in the form of a spiral, adherent posteriorly and laterally, but there was no suppuration. Following this he continued well until October, 1902. At that time there was again an acute attack of typhlitis with no rise of temperature but with great pain over McBurney's point. He was in bed for three months. Subsequently, in March, 1903, another operation was performed and the ascending colon was found to be adherent to the peritoneum. Since that time there have been no attacks, but there is still pain over McBurney's point even upon moderate movement. Moreover the patient still suffers from constipation. During an evacuation, and even for hours afterward, he invariably has great pain in the region of the cecum. The patient reports that since the two operations he is decidedly worse than before.

Examination of the cecal region reveals decided sensitiveness to pressure, particularly in the center of the cicatrix, from the operation; but even the remaining cecal region is distinctly painful upon pressure.

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[1] *Haberer*, "Beitrag zur Appendixfrage, mit besonderer Berücksichtigung von Dauerresultaten." *Arch. f. klin. Chirurgie*, Bd. LXXVI, H. 1 u. 2.

[2] *Leop. Fischl*, "Typhlitis nach Amputation des Wurmfortsatzes." *Prager med. Wochenschr.*, 1904, Nr. 7.

[3] *Gerhardt*, "Perityphlitis mit Rückfällen." *Mittheilungen aus den Grenzgebieten*, 1896, I, p. 354.

Probably in these and similar eases extensive adhesions form whieh result in a more or less decided kinking or stenosis of the ceeum or aseending eolon. As is well known, the prognosis in these cases of adhesions is by no means favorable, and there is no known remedy whieh will positively prevent their rceurrcnee.

Although the number of sueh unfavorable results is relativcly small in proportion to the fortunate sueeesses, ncvertheless they demand our seientifie and serious eonsideration.

From this deseription it is evident that the treatment of chronic perityphlitis must be adjusted to the eharaeter and the nature of its dcvelopmcnt. In genuine chronie perityphlitis it may be expectant and without risk. In the residual forms, with few exceptions, it must be operative. Even in the first group opcrative treatment is the most hopeful. But the indication for this neeessitates most eareful eonsideration whieh devolves ehiefly upon the physieian.

EXAMINATION OF THE FECES

By J. STRASBURGER, Bonn

Until recently but slight interest was manifested in professional circles in the semeiology of the feces and in the question of their diagnostic value. Of course, in the investigation of metabolism, and espeeially of absorption, the chemical examination of the feees has for more than half a century held its place among the methods of the physiologist. But the objects in view had little in eommon with elinical diagnosis.

Omitting from consideration those clinieians who made a speeialty of the study of the digestive organs, the pediatricians were the first to attach importance to the examination of the feees in the differentiation and recognition of pathologieal alterations. The conditions in nurslings are in every respect much more simple and more favorable than in the adult. The dejecta are shown to the physician in the diaper, they have no disagreeable odor, and, on aceount of their simple composition, they necessitate much less diagnostic experienee than those of later life. But this does not alter the fact that also in many diseases of the adult a thorough investigation of the feces is absolutely necessary in order to arrive at an exaet, anatomical, functional, or etiologic diagnosis. I must refer here to Nothnagel's oft quoted statement that in the pathology and diagnosis of intestinal diseases the examination of the dejecta is of even greater importanee than is the examination of the sputum in diseases of the respiratory traet. The foundation for a rational examination of the feces was established by Nothnagel more than twenty years ago in his "Beiträgen zur Physiologie und Pathologie des Darmes." But the last few years only have intensified our interest in this subject, and, fortunately for the patients with intestinal disease, this is still increasing, a fact which is unquestionably due to the labors of Ad. Schmidt.

To investigate the feces sueeessfully and thus to recognize the finer disturbances of intestinal activity we must avoid the difficulties with which earlier investigators were commonly eonfronted. What these were will appear when we ask ourselves why an examination of the excrements of the nursling is so mueh easier and is so mueh oftener earried out than that of the adult.

We have already stated that the infantile feces are always at the disposal of the investigator. If not found in the diaper, we may introduce into the rectum a small instrument something like the bulb of a

syringe, or, still better, an instrument invented by P. Cohnheim—a medium thick glass tube which is inserted within the anus and which, at the end that is introduced, is closed in front; this may readily be inflated ovally. Laterally, at the distended portion, it has an oval opening. If rotated a few times in the ampulla of the rectum we can almost always secure with this small instrument as much material as is necessary for an examination.

We find on estimating the feces of nurslings that the simple and uniform composition of the food, as compared with that of adults, greatly facilitates the process.

The conclusion drawn from this is that we must first make it easy for adults to furnish a specimen of their feces for examination; secondly, in all cases where the change in the dejecta is not so great but that it can be recognized with every kind of nourishment, we must order the patient a definite diet of uniform material. It is evident that deviations from the normal will thus be more readily recognized. With a mixed and constantly varying diet, fecal examination to a certain extent resembles a comparison of two alternating factors which simultaneously influence each other; for the composition of the feces is the result on the one hand of the nature of the food, and on the other hand of the digestive power of the intestine. If we remove from this comparison the one changeable factor, the influence of varying food, and replace it by a constant one, the condition of affairs becomes simpler. In gastric pathology this has been for some time recognized in the form of the Ewald-Boas test-breakfast as well as in the Leube-Riegel test-meal.

But to return to the primary point: In a hospital, naturally, there is no difficulty in procuring the necessary specimen for an examination. But the majority of those who suffer from intestinal diseases do not go to a hospital but to the clinic for treatment. In office practice, however, we must reckon upon the patient's delicacy in regard to everything concerning the dejecta. Here we ask the patients to bring on their next visit a portion of their dejecta, naturally that which appears to them to be pathologic, enclosed in a bottle with a wide mouth, perhaps in a preserving jar with patent lid. Some patients will refuse, for this appears to them to be worse than the disease, and only when they are accustomed to examinations of the kind, or when the physician has the necessary appliances and can furnish them with a glass jar with a proper stopper, will they comply. This proposition, if I am not mistaken, emanated from Raudnitz, and, like many suggestions in practical medicine, is valuable. Pasteboard may be used to place the feces in the glass jar or other container.

The point just discussed is a question of external nature. Much more important and vital is the formulation of a test diet. Based on the previously mentioned considerations, this was introduced into intestinal pathol-

ogy by Sehmidt and myself. At first somewhat eomplieated, it was gradually so simplified by considerations of a seeondary nature that the difficulties connected with it seemed to disappear. We have suceessfully employed this method for a number of years, and have reported the results obtained by it in our monograph upon human feces.[1]

By numerous researches we have, in the main, determined the limits of what may be regarded as normal in a test diet and what belongs to the realm of pathology, and we possess a criterion for the mieroseopic and macroscopic investigation of the feces. Our test diet in the form last advised by Sehmidt[2] is eomposed of the following:

TEST DIET

Morning: 0.5 of a liter of milk (or, if milk is badly borne, 0.5 of a liter of cocoa [made of 20 grams of powdered cocoa, 10 grams of sugar, 400 grams of water and 100 grams of milk]).

With this 50 grams of zwieback.

Midday: 0.5 of a liter of oatmeal gruel (made with 40 grams of oat grits, 10 grams of butter, 200 grams of milk, 300 grams of water and one egg); this mixture should be strained.

Dinner: 125 grams of scraped beef (weighed raw), broiled only slightly so that it is raw in the center, and prepared with 20 grams of butter. With this 250 grams of mashed potatoes (190 grams of potatoes, 100 grams of milk, and 10 grams of butter).

Afternoon: The same as in the morning.

Evening: The same as at midday.

This test diet eontains 120 grams of albumin, 111 grams of fat, and 191 grams of earbohydrates; it corresponds to 2,234 large ealories, and almost furnishes the food requirement of a person not doing eorporeal labor. For the majority of patients with intestinal disease, and with these we are dealing, it is suffieient. Individual caprices of appetite may perhaps be indulged by modifying the quantity of milk without speeially influencing the result of the fecal investigation. The objection has been made that the test diet, on aecount of the unsuitability of milk for intestinal patients, is frequently not effective, and this is undoubtedly true in some cases; but, as Schmidt and I have ascertained by numerous investigations, it applies to only a very small percentage of patients. We should not at once yield the point if the patient states that he cannot digest milk, but should resort to a few well known artifices. As one such, I must mention the avoidance of raw milk. It should be given in the food, and perhaps with various additions. According to Schmidt, 0.3 of a gram

[1] *Schmidt* und *Strasburger*, "Die Fäces des Menschen im normalen und krankhaften Zustande." 2. Aufl., Berlin, 1905.

[2] "Die Functionsprüfung des Darmes mittelst der Probekost." Wiesbaden, 1904.

of pure salicylic acid may be well mixed with some cold milk and added to the entire daily portion of the milk (one and a half liters) and allowed to come to a boil; this is especially beneficial. If it should be necessary, cocoa may be used as a partial substitute for milk in the trial diet.

The test diet might be made up differently, but we prefer to adhere to this form, since, aside from the fact that we have tested its applicability, its modification and further testing would deprive it of its principal advantage which is that after many careful investigations of cases it has furnished us a common basis. Even with patients walking about this test diet may be well employed. I usually give the patient a printed slip with the necessary instructions, and by subsequent microscopic investigation of the fecal specimen he brings with him I am convinced that the directions have been, as a rule, correctly followed. This diet is enforced for about three days, and the dejecta of the last day are investigated. If we desire to be quite certain that the feces are the result of the test diet, or, if it is desirable (and this is important) to ascertain the rapidity of the intestinal passage, the feces may be made distinctive by administering a capsule containing 0.3 of a gram of finely powdered carmine at the beginning of the test diet. This will give to the evacuations a bright red color and the powder irritates the intestine but slightly. If, as sometimes happens, the dejecta in consequence of their own peculiar color do not show this red tint distinctly, the fine carmine granules may be readily recognized under the microscope.

As in all other investigations, fecal examinations for diagnostic purposes should follow a definite plan, and in every case a number of main points be noted. At first we naturally elicit a history of the frequency, the form, and the consistence of the dejecta, their probable admixture, and any abnormal sensations during defecation. I shall not here further discuss the individual points.

Under some circumstances it is desirable to obtain an idea of the duration of the ingesta in the gastrointestinal tract, and this may be readily determined by the administration of carmine at the beginning of the test diet, the results of which the patient may observe for himself.

The order of the actual examinations should be as follows: 1. Macroscopic; 2. Microscopic; 3. Chemical; 4. Bacteriologic.

The macroscopic examination, as must be at once premised, is the most important in practice, but it should be made in a different manner, and much more thoroughly, than is usually the case. As a rule this is followed by the microscopic examination; but, according to the circumstances of the case, or as a matter of choice, the chemical test may be first made, and this should be very simple. The bacteriologic investigation, if not included with the microscopic, is usually unnecessary; it consumes time, and except in certain typical infectious diseases the labor expended upon it is, as a rule, not commensurate with the diagnostic results.

With a flat spatula of wood a portion of the dejecta is taken from the vessel and placed in a large flat glass bowl; we observe the form of the feces (in so far as this is still retained), the consistence, as well as the difference in consistence of the various parts, the color, the odor, and the reaction. The latter is tested with moistened litmus paper, and we may note that the reaction of the surfaee of the fecal mass may be different from that of its interior. Naturally no opinion ean be formed as to the total quantity of the feees except by observation in a hospital of the total amount discharged in at least three days, and passed in a period between the administration of two carmine capsules.

Water is added to the feces in the glass bowl, the masses are disintegrated by the spatula, and if eoarser partieles are admixed these may be extracted with a forceps and placed in a small glass bowl eontaining some water. For this purpose it is best to use two bowls, one with a black, and the other with a whitc lining, and to work in a clear light; Kroenig's table is very convenient (when it is necessary to search for gall-stones, foreign bodies or the like that have been swallowed, all the feces must of eourse be broken up, and for this purpose a feeal sieve which has several times been mentioned should be employed).

A mass of about the size of a hazelnut is plaeed in the mortar, diluted by the gradual addition of water until it has become of a thin fluid eonsistencc, when this is poured into a flat glass dish for further examination.

We now consider the following points, which not only apply to the maeroscopie, but to the mieroscopie and chemical investigations:

1. The remains of food:
   - A. Residue of food, i. e., portions of food whieh even a healthy intestine cannot absorb:
     - (α) The bulk of this is naturally formed by vegetable constituents or more or less enerusted eellulose. In the unopened eells we not infrequently find products which might be serviceable in nutrition, but these are unabsorbed and diseharged in the feees beeause the husks have made it impossible for the digestive juices to reach them.
     - (β) Animal remains: Firm connective tissue, elastie tissue, bone, cartilage, seales of fish, fish bones, hairs, pieces of egg-shell, seales of erustacea and the like.
   - B. True remains of food which are eapable of being digested and may be acted upon by the digestive juices, but for one reason or another have not been absorbed:
     - (α) Remains of albumin, chiefly meat;
     - (β) Carbohydrates, particularly starch;
     - (γ) Fat in different forms.

2. The kinds and the amount of the products of decomposition.
3. Pathologic admixtures from the intestinal wall:
   A. Mucus;
   B. Blood;
   C. Pus;
   D. Remains of tissue.
4. Admixtures of other kinds:
   A. Such as have formed in the body itself (stones, parasites);
   B. Such as have entered the intestine from without (foreign bodies).

Between 1 A and 1 B of this group there is of course no sharply defined line, which is particularly true of a mixed alternating diet with a varying preparation of the individual foods, and this cannot be too strongly emphasized. In our test diet the limitation is much more distinctly drawn.

It is worthy of note that upon macroscopic investigation of normal dejecta no undigested constituents are found which exceed in size the head of a pin. All larger particles, therefore, are of semeiotic significance, and this increases with their size. The conditions are different with a mixed diet. The dejecta of perfectly healthy persons may be well formed and of normal appearance, yet they almost invariably contain the macroscopically visible residue of vegetables, especially fragments of potatoes and turnips, leaves of lettuce, long shreds of asparagus, perhaps even entire lentils, the skins of different berries, as well as their seed. This list might be enlarged at will, and only the dilution of normal feces with water and a cursory examination are necessary in order to find these substances. Diagnostic conclusions as to the function of the digestive apparatus should be arrived at only after careful consideration. It is true we may determine from the presence of such remains that mastication was insufficient and that the food was too rapidly eaten; if these are absent, the digestive organs must, at all events, be in a healthy condition.

Of course, we do not include here the extreme cases in which numerous coarse particles are excreted, especially when the feces have lost their normal consistence, and it is evident at the first glance that these remains of food are but slightly altered, nor do we include cases of lientery. There can be no doubt as to the significance of these signs. Animal food in general, even with a mixed diet, is most easily absorbed. Nevertheless it is not unusual to find shreds of tendons or of arteries, under some circumstances also fibers of meat, and, with a plentiful ingestion, portions of tough or hard unchanged meat.

It is important for the physician to have a clear conception of these facts, since patients, who for any reason begin to observe themselves closely, frequently bring to him for examination some portion of the feces which

causes them anxiety, and the physician should have a thorough understanding of such appearances, particularly if of a vegetable nature. This enhances the wisdom of the physician in the eyes of his patient, who is not only relieved in mind by a negative diagnosis, but desires to know exactly what conditions are present. In such cases it is necessary to regulate the diet of unreliable patients, and the impression is never effaced if

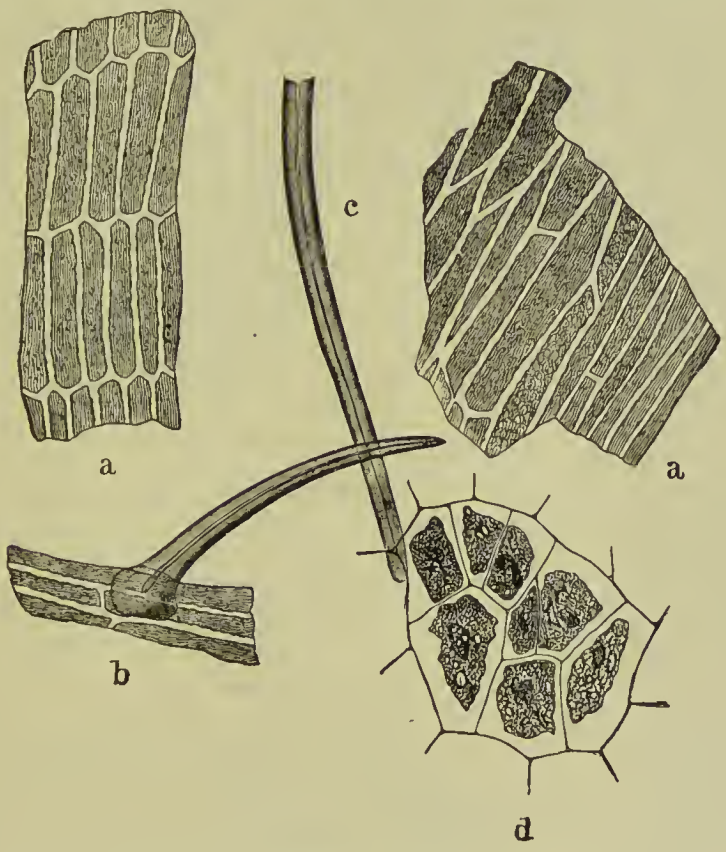

FIG. 22.—A fragment of grain from bread.

a, portions of the pericarp; *b*, portion of the pericarp with the hair; *c*, fragment of a hair; *d*, cells of the gluten layer. · (Magnified 250 times.)

the error in diet is revealed to the patient from the examination of the feces.

I shall first enumerate the food remains which most commonly appear:

While the residua of many vegetables so closely resemble each other after digestion that even the most skilful botanist cannot differentiate them, there are, on the other hand, a number of other food products, particularly from the daily diet, which can be recognized at the first glance. As a rule the aid of a microscope is necessary. A number of illustrations are given which are taken from various dejecta. They have been obtained by centrifugation of the feces rubbed up with water, and by subsequent washing of the sediment, and are much more distinct than the preparations made immediately from the feces.

We seldom fail to find in the feces the remains of grain from bread (Fig. 22). They are the more numerous and the larger, the coarser the bread which was eaten. But these characteristic constituents are also

Fig. 23.—Potato cells.

a, empty, partly contracted and folded; *b*, filled with starchy material. Besides these, a starch granule retaining its external form (stained by iodin). (Magnified 250 times.)

found when only gruel has been eaten: portions of the chaff and the hulls of the seed, hairs from the epidermis, and fragments of the gluten cell layer.

Almost as frequently as remains of grain we find large transparent cells from the parenchyma of potatoes, and these become conspicuous on even slight enlargement (Fig. 23).

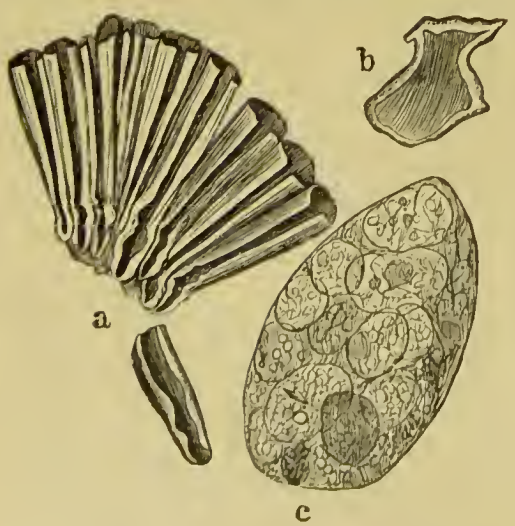

Fig. 24.—Fragment of peas.

a, palisade cells of the seed hull; *b*, a carrier cell of the seed hull; *c*, a cell from the parenchyma of the germ leaf filled with gluten and starch. (Magnified 250 times.)

These shreds are very distinct, and upon the addition of Congo red to the preparation, other delicate vegetable tissues are also revealed. As a rule they are empty and pressed flat; but some of them contain agglutinated starch, that is, dextrin, and in this case, if a sufficient quantity of Lugol's solution is employed, they change from blue to red.

The ingestion of beans, peas and lentils may be demonstrated very easily (Fig. 24).

Every preparation will reveal many glistening, greatly thickened structures in columnar form, the

so-called palisade cells. Resembling these, but broader and much shorter, are the carrier cells. In peas these are detached from one another, and in the microscopic preparation they are always isolated. The carrier cells of beans, however, form a continuous layer of polygonal cells which, viewed from the surface, resemble a carpet. Each of these cells contains a crystal of calcium oxalate. The previously mentioned parts belong to the hull enclosing the seed. The large round cells resembling potatoes filled with gluten and starch granules form the parenchyma of the germ leaves.

Fig. 25.—Carrot cells with crystals of carrotin. (Magnified 250 times.)

In the preparation (Fig. 25) we note very pale, large cells which contain small, isolated structures, orange to fiery red in color (carrotin), partly crystalline, partly of more irregular form; a finding which is very conspicuous and denotes the presence of particles of carrot.

The ingestion of various leaf-like vegetables is recognized chiefly from the delicate epidermis with wavy cell outlines showing intermediate spaces,

Fig. 26.—A portion of the leaf of a head of lettuce. Above, the epidermis with wavy limited cells and with spaces; below, the chlorophyl granules in round cells and a branching bundle of vessels. (Magnified 250 times.)

Fig. 27. — Two stippled vessels in juxtaposition. (Magnified 250 times.)

the chlorophyl granules and vessels running in the ribs of the leaf serving for the conduction of water, and adorned with thickened rings and with spirals (Fig. 26).

These spirals (see Fig. 27) are found less abundantly in other edible parts of vegetables, such as the stems and roots, and are less developed in the delicate leaves of flowers and fleshy fruits. They become tough and consequently so indigestible that after the consumption of even small quantities of these vegetables they invariably appear in the preparations. Often the spirals are drawn apart, or are irregularly layered among each other, or are mixed with animal fibers into a mass which cannot be separated.

Fig. 28.—Elongated cells from the stalks of asparagus. (Magnified 250 times.)

In the next preparation (Fig. 28) we see elongated, thin-walled cells originating from a fiber of asparagus. These long threads may be observed even macroscopically in the feces; they are usually attached to shreds of tissue or the hulls of peas and beans with which they may be confounded. The microscopic picture is shown because upon superficial view there is a decided resemblance to the mucus of the feces, as may readily be seen later. Still, the more clearly outlined circumference is conspicuous. If we find the so-called raphides, parallel bundles of fine needles consisting of calcium oxalate, this is additional proof of the vegetable nature of the preparation. The characteristic cellular inclusions which are noted in intestinal mucus are, of course, lacking.

Fig. 29.

Fig. 30.

Fig. 29.—Spores of truffles in an ascus. (Magnified 250 times.)

Fig. 30.—Mildew spores of wheat (tilletia-caries), producing stone or stinking rust. (Magnified 250 times.)

Fig. 31.—Spores of lycopodium clavatum (semen lycopodii). (Magnified 250 times.)

The dark truffle spores which appear in the feces after partaking of this food are found in several combined transparent colorless tubes: they have fine thorns (Fig. 29) and are easily recognized. The same is true of the spores of ustilagineæ of grain (tilletia-caries) (Fig. 30) which not infrequently are found in flour. A knowledge of these is of importance, since they may appear to the novice to be the ova of intestinal parasites; for instance, of ascaris lumbricoides.

Above all, they are decidedly smaller. The same error may be made with lycopodium seeds (Fig. 31) which, as is well known, are often used as a coating for pills. They appear to be partially consumed in digestion.

As the remains of fruit, I shall mention only the very characteristic core (stone cells) of pears which gives to the pulp its toughness, and which is present even in the finer varieties (Fig. 32).

The possibility of confounding the tubules of oranges with intestinal parasites must be pointed out to the investigator, especially if he limits himself to a macroscopic inspection. Virchow was the first to describe a case of this kind.

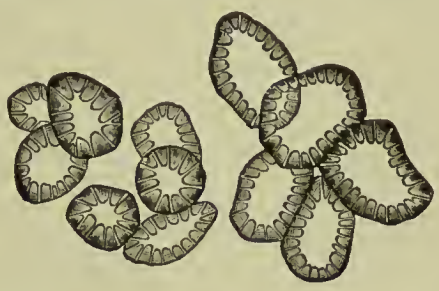

FIG. 32.—Stone cells (core) from the pulp of pears. (Magnified 250 times.)

I must devote a few words to the elastic fibers in the remains of animal food. These belong to the parts which are difficult to digest but not wholly indigestible; their dissolution may occur in the gastric juice as well as in the pancreatic juice. With a mixed diet we frequently find elastic tissue, and attach no especial diagnostic importance to its appearance. Sometimes we observe its well known double contours, beautifully wound fibers in an unchanged condition, but their continuity somewhat broken by the digestion of the intermediary tissue

FIG. 33.—Elastic fibers. (Magnified 250 times.)

(Fig. 33); sometimes we see them in a half digested condition, traversed horizontally by numerous lines so that they may be confounded with fibers of wool.

A middle position between the residua of food and the true food stuffs may be assigned to connective tissue (Fig. 34). The form in which it is ingested, more than the absolute quantity, accounts to a great extent for its reappearance in the feces. According to Schmidt the greatest bulk of connective tissue is left by smoked meat, next by raw meat, still less by fried meat, and least by tender boiled meat. Consequently, especially after the ingestion of raw ham, we not infrequently find enormous amounts of connective tissue convolutions which permeate the feces like a fine film, and which appear only upon breaking up and washing the dejecta with water.

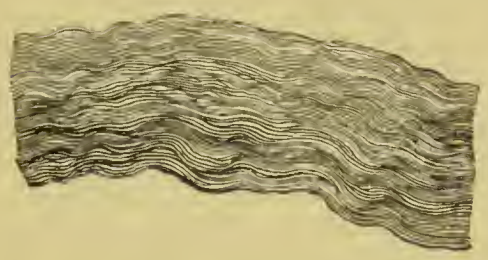

FIG. 34.—Connective tissue. (Magnified 250 times.)

The knowledge of this fact warns us to be less liberal in allowing patients raw ham than is the case at present.

We see that with a freely chosen diet the presence of connective tissue in the feces as a rule permits no conclusion as to the function of the digestive apparatus, provided large quantities have not been passed. The circumstances are different with our test diet, and upon this foundation Schmidt formulated a very valuable clinical test. Raw connective tissue, for instance, is digested only by the gastric juice, not by the pancreatic juice. Now the test diet contains 125 grams of raw scraped beef which, in order to make it palatable for those patients who refuse to eat raw meat, is broiled very lightly on the outside. Experience proves that the connective tissue in this so-called "German beefsteak" is under normal circumstances so completely digested that macroscopically no residue can be found in the feces.

Even the smallest portions visible to the naked eye point to a disturbance of digestion which, as is obvious from the preceding, has its seat in the stomach, since only this organ has the power to digest raw connective tissue. As a rule, in such cases there is a decrease in the secretion of hydrochloric acid. Under some circumstances, however, other disturbances of the gastric function are at fault. Thus the investigation of the dejecta may in certain cases point to an unsuspected gastric affection, and under others may give us a clue to the malady even when the patients refuse the introduction of the gastric tube for diagnostic purposes.

We shall now consider the manner in which the true remains of food appear in the feces, these being chiefly represented by muscle fiber, starch and fat, the latter in varying chemical combinations. From the shape and quantity we endeavor to form an opinion as to the function of the digestive apparatus, especially of the intestines and the large contiguous glands, the pancreas and the liver. We can then decide in how far a freely chosen

diet is permissible, and in how far we may succeed with the test diet proposed by Schmidt and myself.

As a rule remains of meat, even with a mixed diet, cannot be found by microscopic inspection of the feces. Therefore, the presence of large portions must be regarded as pathologic. But in the test diet the limits are more sharply drawn. Here the presence of the smallest portion of muscle, detected without magnifying, denotes disturbance either on the part of the small intestine or of the pancreas, while an affection which implicates only the stomach or the colon does not cause the excretion of macroscopically recognizable remains of meat. We may therefore to some extent form an opinion as to the seat of the disturbance of function. It is true this gives us no deep insight into the *nature* of the affection. The small intestine and the pancreas have in common, as is well known, three important functions, secretion, absorption, and motility. It would be a great advantage if we could recognize in the individual case which of these factors has suffered. As a rule, however, it is impossible to form any opinion in regard to this. A decrease in the digestion of meat, taken by itself, is no certain guide.

If the excreted fragments of muscle are found permeated by connective tissue it is obvious, from what has been previously stated, that a faulty function of the stomach is present besides the intestinal disturbance.

In the microscopic discrimination of muscle fibers we must be much more cautious than in the macroscopic recognition of residue of meat in the excretion. In man, after the ingestion of meat, we invariably find small fragments of muscular fiber in the feces, and according to the stage of digestion in which they are found three varieties may be differentiated: 1. Large, distinctly transverse, striated shreds with sharp, angular contours; 2. Medium sized particles with rounded edges, their transverse striæ less distinct; therefore a well-developed longitudinal striation is usually visible; 3. Small polygonal or round cells whose striation has been somewhat obliterated or is entirely lacking (Fig. 35).

Fig. 35.—Muscle fibers in various stages of digestion. (Magnified 250 times.)

Between these three forms there are, of course, many transitional stages. After administering a test diet we may assume an intestinal disturbance if many remains of muscle of the first mentioned category are conspicuous in the preparation. In such instances, as a rule, fibers of meat may be recognized macroscopically.

From the foregoing it is obvious that a more accurate method of esti-

mating the amount of meat digested is a practical necessity to enable us to form a definite opinion regarding this function of the intestine. The first improvement in this direction we owe to Kermauner. But his method, based upon counting the number of musele fibers in a microscopic preparation, is laborious and does not always ensure aecuracy. The method elaborated by Schmidt is better. In this the amount of the eleansed sediment of the feces is determined volumetrically, after which it is again subjected to digestion. By this process all the residue of meat which is at all digestible is acted upon by the digestive juices and dissolved. If the remaining volume of sediment is then deducted from that first found, we have the actual amount of proteids which are insusceptible to intestinal digestion. In employing the test diet, as Sehorlemmer ascertained under Schmidt's direction, the difference between the normal and pathologic intestinal funetions may very easily be determined. This may be utilized in scientific researches, but for clinical praetiee it is mueh too laborious to find wide application.

Until very recently, the question has been much discussed whether, and in what form, *starch* normally appears in the feces. In order to form an opinion, we must first of all separate the starch granules or agglutinated shreds which are enclosed in cells from the free starch. With a mixed diet the first forms are invariably found in the sediment of the feces. Nevertheless Nothnagel's opinion that *perfectly retained free starch granules* are normally absent is correct. Naturally we must be careful to see that starch is not liberated during the preparation by pressure upon the cells; for this reason, in the question under discussion, weight is attached only to the opinions of those authors whose researches into the dejecta have been characterized by great precision and the consideration of this point. The reports of Ledden-Hulsebosch, whose investigations were marked by extraordinary attention to detail, are especially reliable. Ledden-Hulsebosch found that even if well retained granules do not appear with a mixed diet, nevertheless agglutinated starch in the form of layers and shreds lying free is detected. Naturally this can only be determined by the careful investigation of the cleansed sediment of the feces. The addition of iodin to the simple microscopic preparation, even if a large amount is added, frequently produces no result, although it can be certainly proven by other methods that starch is present. This certainty is attained if we subject the feces to a "fermentation test," or, as Schmidt generally expresses it, to an "incubation oven test"; i. e., the feces diluted with water in a suitable vessel are subjected to the heat of an incubation oven after which we note whether processes of decomposition appear and also their action. If the absorption of food has been so complete that the feces show in the main only a residue, no decided change takes place after about twenty-four hours in the incubation oven. If, however, constituents of the food which may be readily acted upon are admixed (and this is par-

ticularly true of albumin, starch, and to a less degree of fat), these are broken up by the microörganisms of the feces on the formation of the characteristic products of decomposition. According to the character of this process, we differentiate between fermentation and decomposition.

Fermentation, as a rule, runs a more rapid course than decomposition, and is attended by the production of odorless gas and an increase in the acidity of the feces, the color of the dejecta becoming lighter. The process of fermentation depends upon the decomposition of carbohydrates, particularly of starch, which is changed into sugar by the diastatic ferment always present in the feces, and is further disintegrated by the bacteria present in large amounts. *Decomposition* is slower; gas, as a rule, is developed in less amount but is characterized by its disagreeable odor. In this process the feces become alkaline, their color darker. This is the consequence of decomposition of albumin, and less of the remains of food which do not so readily undergo decomposition, as, for instance, the pathologic products which are excreted from the intestinal wall, such as mucus, pus and blood.

A positive reaction of the fermentation test, therefore, shows us the presence of carbohydrates in the feces. A point to be observed is whether they are of such nature that they may be readily acted upon by the bacteria, and are therefore susceptible to intestinal digestion and, with sufficient power of the intestine, would have been digested. We are dealing with actual *food residue,* not the remains of food. In accordance with the fact previously mentioned that we frequently find free remains of starch, the fermentation test with a mixed diet is often positive. It is a fine reagent, and gives results even in cases in which the ordinary examination, the usual microscopic investigation of starch, is unsatisfactory. If the amount of carbohydrates in the food is limited to a certain extent, as in our test diet, fermentation will normally not be present, as the intestine is fully capable of absorbing the amount of carbohydrates it contains. In some cases, however, even after the test diet, the feces contain starch and ferments; we are then justified in speaking of the "insufficiency of starch digestion."

At the same time, the other foods may be well absorbed. Such an occurrence is not rare, and it forms a symptom-complex which Schmidt and I have described as "intestinal fermentative dyspepsia." In this condition, so far as our observations extend, the stomach, colon, and probably also the pancreas, function in a normal manner; since the labor of starch digestion is laid principally on the small intestine, we have reason to believe that we are dealing with a disturbance of the small intestine, probably with an anomaly in the secretion of the intestinal juice, a secretion which has normally a powerful amylolytic action. To demonstrate fermentative dyspepsia while employing the test diet, we use

the fermentation test, a method which, as we learn by experience, is superior to searching the microscopic preparation for starch.

I shall demonstrate from the feces of a patient the relation to intestinal fermentative dyspepsia. These dejecta were from a powerfully built, fairly well nourished man in middle life, who for months had complained frequently of abdominal pain particularly in the region of the navel, also of a certain lassitude and malaise. There were no gastric symptoms. From time to time there was constipation; frequently, however, there were two or three thin, pappy stools daily. Actual diarrhea was never present. Upon careful questioning the patient recalled that the dejecta were sometimes foamy, of a light color, the odor sometimes resembling that of old cheese. On such days the fecal evacuations occurred three or four times in twenty-four hours. The consistence, however, was not fluid but always pappy. External examination of the chest and abdominal organs revealed only a moderate distention of the abdomen and a diffused sensitiveness to pressure around the navel, particularly to the left. The epigastrium was not painful upon pressure; the tongue was slightly coated. An examination of the evacuated gastric contents showed them to be normal. The feces were then examined, as it was to these the history pointed.

The patient was instructed to bring a specimen of the dejecta for examination, but I shall not relate what was then found as the investigation revealed nothing definite. The man was then placed upon our test diet. On inspecting the feces it was noted that the consistency was pappy, somewhat thinner than normal, the reaction moderately acid. Otherwise both microscopically and macroscopically no abnormal admixture was found. Meat and fat were well absorbed. On the other hand, the number of potato cells was conspicuous; most of them were empty; with iodin they occasionally revealed demonstrable starch. No free remains of starch were found. The fermentation test was then made in the following manner:

About 5 grams of the well mixed feces were disintegrated by means of a suitable instrument (a wooden spatula). If the feces are hard less should be taken, if thin a correspondingly larger amount, so that approximately about the same amount of dry substance is worked up. The fermentation vessel [1] (Fig. 36) is taken apart so that only three rubber plugs are combined with one another. In *a*, the lower chamber of the fermentation tube, the feces are stirred up with water, after which the rubber plug is placed within the tube to prevent the formation of air bubbles. The chamber, *b*, is filled with water and also closed with a smaller rubber plug, so that no air bubbles can form in the tube. Finally, the tube, *c*, which has at its apex a small hole for the escape of air, is placed in position. The illustration shows how far the glass tubes which are united

[1] Made by C. Gerhardt in Bonn.

project into the lumen of the larger tube. After the parts of the apparatus are attached, it is placed for twenty-four hours in an incubation oven heated to 37° C., or into a water bath of the same temperature. If gas develops from the feces, a corresponding amount of water is forced into the ascending tube, *c*, where the height of the water may be read off. The reaction of the feces should be tested with litmus paper before and after fermentation. The diagnostic employment of fermentation is only indicated after the test diet recommended by Schmidt and Strasburger.[1]

Of course, any other fermentation apparatus may be employed; for instance, those used in the examination of urine. Our fermentation tubes, however, permit a more accurate opinion as to the amount of gas formation since nothing is lost in fermentation, and the amount of displaced water may be more easily read than that of the gas which has been formed, since this in part permeates the feces in the form of bubbles.

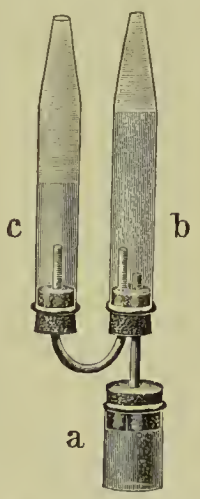

FIG. 36.—Fermentation tubes with contents.

Positive reaction of the fermentation test. The quantity displaced in tube, *c*, is greater than that of the gas in the upper part of tube, *b*, as a portion of the gas has been retained in the feces in the vessel, *a*. (One-fifth of natural size.)

The test is to be looked upon as positive when at least a third of the tube, *c*, is filled with displaced water. This happened in our case. The examination with litmus paper showed a positive acid reaction of the fermenting feces, and we then determined that the gases formed were odorless. In the patient in question we could scarcely have formed an opinion as to the malady present except by this test. It is true our diagnosis is a purely functional one, the anatomical disturbance causing it being still unrecognizable. But in the great majority of cases it is probably due to catarrh of the mucous membrane of the small intestine.

By the aid of the fermentation test we have come to a conclusion as to the nature and the degree of the processes of decomposition to which the feces, if we may be permitted the expression, are predisposed. In well developed cases this decomposition attains its full development in the intestine itself, and the dejecta when discharged are either light, foamy, and acid, the odor reminding us of butyric acid, or they are dark, have an alkaline reaction and a fetid odor, which, under some circumstances, may be even putrid. In comparison with the preceding, the disturbances last enumerated are always more serious, and correspond with what we learn from the subjective examination of the patient.

I have included in my discussion of the absorption of albumin and

[1] Compare *Schmidt* and *Strasburger*, "Die Fäces des Menschen." Berlin, Hirschwald, 2. Aufl., 1905; or, *Deutsches Archiv für klin. Medicin*, LXIX, p. 570.

carbohydrates the description of the processes of decomposition, but I have not yet touched upon the question of the digestion of fat, which naturally must be included with that of albumin and starch, and which we shall now consider.

Fat is found in the feces in different forms; in the adult chiefly as soaps of earthy alkalies, in short colorless crystals and needles, or as flakes. The latter are either colorless or tinged yellow to brown by biliary coloring matter when they form the yellow calcium salts, so called by Nothnagel. More rarely we observe free fatty acids, long curved needles such as are also observed in the sputum in putrid bronchitis. Neutral fat, according to its melting condition, occurs either in the shape of granules or drops. The latter are normally absent in adults on account of the ready absorption of fats that are melted. On the other hand they may be found in healthy nurslings. In the microscopic preparation they form structures with borders, like the lakes upon a map, and are stained an intense yellow by bilirubin.

What form of fat is present is generally very easy to decide: Upon heating of the preparation neutral fats and fatty acid needles coalesce, and may be separated by solution in ether. In the soaps this is possible only after splitting the preparation with heat and adding acid. This reaction will also give us some idea of the number and size of the fatty acid drops in the quantity of fat present in the preparation. In Fig. 37 we see, highly magnified, the many soap needles of a fatty stool, while Fig. 38 shows the same preparation after heating and the addition of dilute sulphuric acid. I chose this acid chiefly because the calcium crystals so numerously present in the preparation furnish proof that the base of the soaps with which we are here dealing is principally calcium. This metal combines with sulphuric acid and forms calcium sulphate. Such calcium soaps, when mixed with fibers of vegetables, particularly after the ingestion of olive oil or after oil enemata, may be found in the feces, often as concrements which are erroneously considered to be gall-stones. A quack in this neighborhood utilizes this fact in the treatment of many patients who come to him with various symptoms; he institutes an oil treatment for gall-stones, and at the conclusion of the treatment he hands to the patient the "stones" which have been passed. It is true that, after a little time, these stones crumble in the hands of the patient.

Of course a considerable increase of fat in the stool may be easily recognized. If, however, the amount of fat is less marked it is often difficult to define the limits of what may be regarded as normal. Only exact chemical analyses, too intricate for ordinary practice, will enable us to arrive at conclusions. Upon microscopic investigation, however, we may assume an increase when in nurslings (except very young breast-fed children) flakes of fat are found profusely permeating the entire preparation, and especially if free fatty acids and soaps are also found

in large amounts. Drops of neutral fat are not normally present in the feces of adults except after the administration of castor oil. They are, however, observed after severe diarrhea, and then are stained an intense yellow by bilirubin. The appearance of numerous fatty needles, too, is pathologic. In jaundice, for example, the feces consist almost wholly of soap needles. On employing our test diet we find, as Schmidt determined, that only soaps in the form of unstained flakes or as yellow calcium salts are normally present; there are neither fatty acids, nor soap needles, nor neutral fat drops.

The presence of abnormal quantities of fat in the feces may be attributed to very different causes: To the absence of bile, to disturbance of the pancreatic secretion, or to disturbance of intestinal digestion. In the given case it is by no means easy to apprehend clearly which of these factors is present. In the examination of the feces, more than in any other way, we are impressed with the absolute necessity, for purely diagnostic reasons, of instituting various chemical tests which are sometimes not at all simple.

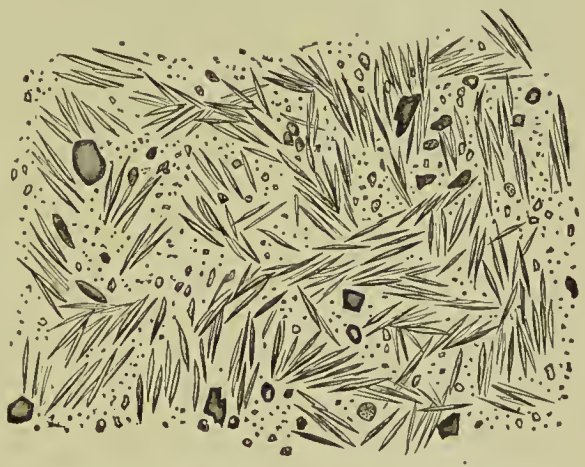

Fig. 37.—Soap crystals and soap granules. (Magnified 500 times.)

I shall illustrate this by a concrete case. The feces, which were of a salve-like and uniform consistence, and of silver gray or whitish color, had but slight odor, and were of moderately acid reaction. They appeared to show the characteristics of the feces in occlusion of bile. The fact was therefore surprising that the patient who evacuated these feces showed no sign of jaundice, and passed clear urine which was free from biliary coloring matter. An occlusion of bile, therefore, was not present, and the following possibilities were suggested and an attempt made to clear up the diagnosis by further investigation. The lead color of the feces might be due: 1. To a cessation of the secretion of bile; 2. To a too great reduction of hydrobilirubin, thus forming leukohydrobilirubin; 3. To an immoderate amount of fat which, under some circumstances, is alone sufficient to cause the ashy color of the feces.

From the salve-like consistence and the peculiar luster of the feces in our case we decided upon an increase of fat. The microscopic preparation showed numerous soap needles, and upon heating and adding acid, large numbers of acid fat globules were formed. The preparations (Fig. 37 and Fig. 38) which have been already described were obtained from these feces. The marked increase of fat in the dejecta was therefore

positive, and it is quite likely that this accounted for the peculiar color of the feces. We then instituted another test, an attempt to prove the presence of biliary coloring matter. For this purpose I mixed a large portion of the feces with dilute acid, heated it to split the soaps, and after cooling I added ether. Thus it was possible to remove a portion of the fat, after which the color of the remaining feces became a distinct brown. I subsequently attempted to prove the presence of biliary coloring matter, and for this purpose added to the fresh feces some hydrochloric acid-alcohol. When hydrobilirubin or leukóurobilin (bilirubin is not found in the normal feces of the adult) is present, the coloring matter may be obtained from the feces in the following manner: To the clear filtrate of the extract, I add a few drops of ammonia and a solution of zinc chlorid, whereupon, after some time, and in a clear light, a beautiful greenish fluorescence is noted. Leukohydrobilirubin, which is demonstrated by this test, is changed back into hydrobilirubin. Another portion of the extract I subject to a spectroscopic examination. With a suitable dilution the characteristic absorption bands are found to be very distinct in the markedly broken portion of the spectrum, between the lines *b F*, which move somewhat to the left when the fluid is rendered alkaline. This, therefore, furnishes proof that the biliary coloring matter has been excreted and has reached the feces. That a too great reduction of coloring matter was the cause of the white color of the feces as well as their richness in fat is improbable on account of the great amount of fat present in the stool, for the presence of leukourobilin does not normally cause a disturbance in the digestion of fat.

Fig. 38.—The same preparation as Fig. 37, but after heating with dilute sulphuric acid.

The fatty acid is seen in drops. In the interior and at the periphery of a large drop, fatty acid needles are beginning to crystallize. The large crystalline needles are calcium crystals and prove that the base of the soap is calcium. (Magnified 500 times.)

The whitish appearance of the stool is therefore due to the immoderate amount of fat present, and the question now arises to what the insufficient absorption of fat is to be attributed. As the absence of bile cannot be considered the cause, the possibility remains of a disturbance in the secretion of the pancreatic juice, or a disturbance of intestinal digestion. The examination of the feces also gives us many points which aid in the elucidation of this question.

Absence of the pancreatic secretion often leads to a deficient splitting

of fat, which is excreted in the feees chiefly as neutral fat. Only the most minute quantitative ehemieal analysis will positively demonstrate this. Nevertheless if numerous soap crystals are present when we examine the mieroscopic preparation, or, on the other hand, nentral fat appears in the form of drops, we may form a definite opinion. Notwithstanding decreased fat splitting, the total quantity of fat excreted is not neeessarily increased; but, besides a sufficient splitting of fats, a large amount of fat may be observed in the feees in disease of the pancreas. The picture therefore varies greatly.

The absence of the pancreatie secretion also causes, as a rule, a decided decrease in the absorption of albumin which may be recognized in the mieroseopic preparation by the presence of numerous fragments of musele with sharp edges.

Ad. Schmidt has called attention to another significant feature. It depends upon the fact that cell nuclei are digested only by the panereatic juice. If this secretion is absent the cell nuclei are found, for example, in the musele fibers. Sehmidt considers it advisable to give small pieces of meat which have been hardened in alcohol, and subsequently placed within very thin gauze coverings. These are easily detected in the feces, and if in natural condition, otherwise after staining, they are examined for the presence of eell nuelei. This test can be utilized only if the result is positive, since under some circumstances intestinal deeomposition alone is sufficient to destroy the cell nuclei.

In doubtful cases the investigation of the feees after the administration of pankreon may give us light.

Other tests for the diagnosis of disease of the pancreas must be omitted since they do not pertain to our theme. But a point to be specially emphasized is that the symptoms of disease of the pancreas appear, as a rule, only when the gland is nearly or entirely destroyed. They may be absent even with complete occlusion of the excretory ducts, and this leads us to think that the secretion may find its way into the intestine by some other channel. Henee mild disturbances of the panereatie function invariably escape recognition.

If, in diseases of the pancreas, the increase of fat in the dejecta is due to intestinal distnrbances, we often find an increase also of the remains of meat, but in this case it appears to be less marked. As the excretion of fat in intestinal diseases is chiefly caused by deficient absorption, eventually other signs of a lack of absorption appear, and confirm the diagnosis.

The presence of dissolved albumin is especially important because this is not found in normal feces. Great care is necessitated in its demonstration, as we have learned by the experiences of the last few years. It is espeeially important not to confound it with the nueleo-proteid of the feces which is invariably present. The easiest and most reliable method

appears to be that of O. Simon[1]: Water is poured over the fresh feces and the mixture is allowed to stand for some time, after which the liquid is carefully drained off and filtered. To make it perfectly clear diatomaceous earth is added, the mixture is shaken, and again filtered. Dilute acetic acid is then cautiously added to precipitate the nucleo-proteid, and the mixture is again filtered. It is difficult correctly to estimate the amount of acid necessary, for if the acetic acid is in excess it will again dissolve the nucleo-proteid. If, after the addition of acetic acid, a slight turbidity appears which is very difficult to filter, another acetic acid test may be made with larger amounts, and subsequently a test for albumin with dilute potassium ferrocyanid solution. The precipitate which appears with this must be decidedly more marked than in the first test if it is to be utilized as a proof of the presence of albumin (native proteid bodies and albumoses).

We can also attempt to demonstrate albumin in a watery extract of the feces by the aid of the biuret test (caustic potash and dilute copper sulphate). This is, however, often difficult because the coloring matter of feces (hydrobilirubin) reacts with the biuret test just as it does with albumin. On using the simple methods which are employed to remove the coloring matter a portion of the dissolved albumin bodies is also lost; they are usually present in only very small amounts; Ury's[2] process, which is the most accurate one, is much too difficult for the general practitioner, especially as it necessitates the removal also of the nucleo-proteid of the feces.

Leaving the discussion of Simon's test for dissolved albumin, I must return to the investigation of the feces under consideration, since it was demonstrated that they contained dissolved albumin in considerable amount. The signs previously enumerated as favoring panereatic disease were entirely absent.

The result of our investigation therefore showed that the marked increase of fat in the feces was due to the cessation of absorption in the intestine. The question then arises, What is the cause of this disturbance in absorption? Here, however, I cannot minutely discuss this question, for the investigation detailed gave no further results, and it is my primary object to describe a number of methods of investigation and diagnostic considerations which may be utilized when the feces are difficult to analyze.

When describing the methods of demonstrating biliary coloring matter I omitted to call attention to a test which is of general application, and in a diagnostic respect leads to important conclusions. This is the corrosive sublimate test invented by Ad. Schmidt, to which a few words will be devoted. This test shows us by a color reaction whether the mass of feces or any isolated portion of the same is colored by bilirubin or by its reduction product, hydrobilirubin. The former is changed to green by the

[1] *Archiv f. Verdauungskrankheiten*, 1904, IX, p. 197. [2] Ibid., Bd. IX, 1903, p. 219.

corrosive sublimate test, the latter to rose red. We now know that the contents of the small intestine are normally stained green by the corrosive sublimate test, those of the colon red, and that the ileo-cecal valve forms a sharp boundary. This is due to the fact that below this region the processes of decomposition set in which, hand in hand with reduction, transform all bilirubin into hydrobilirubin. Only in the nursling, in whom intestinal decomposition is normally absent or very slight, are the conditions different, and here the coloring matter of the feces wholly or in part is composed of bilirubin. If we find unaltered biliary coloring matter in feces other than those of nurslings, it is obvious that

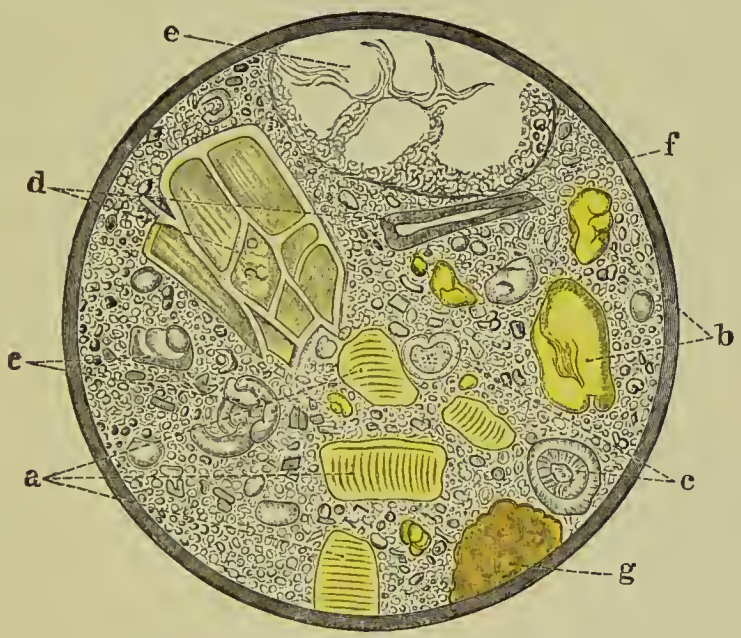

FIG. 39.—Normal feces from a test diet.

a, muscle fiber remains; *b*, yellow calcium salt; *c*, soaps; *d*, chaff remains; *e*, empty potato cell; *f*, detritus; *g*, cacao remains. (Combination picture.)

the coloring matter is from the small intestine or the upper part of the colon, and in consequence of too rapid propulsion or other less obvious reason it has permeated the feces unchanged. At all events we are dealing with a disturbance which, as a rule, may be referred to the small intestine. The examination of the feces for residue of meat, the results of the fermentation test, the characteristic mucus particles, will then serve to localize the affection. If the feces contain much bilirubin they will be of a bright, golden-yellow color, like the feces from healthy, breast-fed children. This is found in the rare cases designated by Nothnagel as jejunal diarrhea. More frequently isolated fragments, particularly of muscle fiber and mucus, show a greenish discoloration with corrosive sublimate.

To carry out Schmidt's corrosive sublimate test a portion of recently evacuated feces, the size of a hazelnut or walnut, is carefully diluted with

a considerable quantity of a concentrated bichlorid solution. This mixture is permitted to stand for several hours or a day in a broad, flat glass dish with a cover. The time consumed is, of course, an objection to this method. The green color of the familiar Gmelin reaction immediately shows the presence of bilirubin. Unfortunately, however, it cannot be used for the finer tests. If bilirubin is present only in microscopic amounts, Gmelin's test is unreliable.

Before concluding this discussion of the remains of food in the feces, I wish to call attention to two illustrations, after Schmidt, of the "test

FIG. 40.—Pathologic constituents in feces after a test meal.

a, large fragments of muscle; *b*, needles of fatty acids and soaps; *c*, neutral fat; *d*, starch granules in a potato cell; *e*, granulose containing bacteria; *f*, yeast cells. (Combination picture.)

of the function of the intestine by means of the test diet." In a combination picture they exhibit the constituents of the feces after a test diet, the first illustration showing normal, the second, pathological conditions.

A large proportion of the total fecal mass is formed by bacteria; this is at once corroborated by the investigation of a microscopic preparation. A large portion of what is otherwise designated as detritus proves, upon close investigation, to be more or less well preserved microörganisms. By a special process I mechanically separated these from the other constituents of the feces, and by weighing ascertained that with an easily digested diet bacteria formed about one-third of the total amount of solids. Upon an average 8 grams of dried bacteria are passed during twenty-four hours. An estimation of their number based on this weight and upon the medium size of the bacterium coli communi gave the extraordinary figure of about

128 billions, a sum absolutely beyond our powers of conception. In this article I cannot enter more minutely upon the subject of fecal bacteria. So far as the familiar pathogenic agents which find ingress by way of the intestine are concerned, they have been referred to in special articles. The question of the importance of the other microörganisms in the feces is, however, much too complicated to be briefly considered. With the exception of the bacteriology of the feces of nurslings, the diagnostic conclusions reached have been comparatively few.

Nor can I devote much attention to the description of the animal parasites. (See volume on "Diseases of Metabolism and of the Blood, Animal Parasites, and Toxicology.")

In a diagnostic respect, the demonstration in the feces of certain pathologic products secreted by the intestinal wall is very significant. In point of frequency, this is especially true of mucus and of blood. The presence of pus or other tissue constituents is much more rare.

Mucus is found in the feces in varying form and distribution. Sometimes it is intimately admixed; this occurs only with a pappy or fluid consistence of the excrement, and, under some circumstances, the flocculi are then so small that they cannot be readily recognized; at other times coarse shreds which originate from the grooves in the haustra of the colon are adherent to the surface of the feces, which are frequently hard. Very dry dejecta are occasionally coated with a thin film of mucus resembling a varnish. Excrement which consists exclusively of large mucus masses, perhaps in the form of tubular casts of the intestine, is found in enteritis membranacea or in colica mucosa. Less massive quantities of hemorrhagico-purulent mucus are voided by patients with dysentery associated with marked tenesmus. The mucus in the feces is sometimes glassy and transparent, sometimes very turbid or even white, according to its impregnation with fat and its permeation with cells. Its consistency varies from that of extreme fluidity and elasticity to the toughness of thin leather. When larger amounts are evacuated confusion with animal membranes or connective tissue convolutions from the food, or even with animal parasites, is quite possible.

Fig. 41. — Connective tissue from the feces. (Natural size.)

The first illustration (Fig. 41) upon a black base shows a coarse shred of connective tissue detached from the feces and washed clean, and beside it a mucus shred of the same size (Fig. 42). The latter differs from the

white connective tissue by its dirty color; the individual processes are not so sharply defined; they are more rounded, and in the thin portions are somewhat transparent. In doubtful cases a microscopic examination must always be resorted to, and this will at once enable us to form definite conclusions (compare Figs. 43 and 44).

Fig. 42.—Mucus from the feces. (Natural size.)

The small, transparent, mucus flocculi which are most common are best recognized when the feces are thoroughly diluted with water and spread out in a thin layer upon a glass plate which is lighted from underneath, or in a glass vessel which is held toward the light, the feces then being permitted to trickle down the sides of the vessel. Mucus flocculi may then be noted as circumscribed transparent particles. Even with these precautions mucus may be confounded with swollen fragments of undigested vegetables or fruit (for example, potatoes, carrots, pears), and microscopical proof should always be obtained since this leads to definite conclusions.

A feature of mucus to be recognized microscopically is the delicate, often scarcely visible, contour, as well as a number of irregular lines in groups and running almost parallel (Fig. 43). After the addition of dilute acetic acid to the preparation and a thorough mixing, the precipitate of mucin causes a reaction characteristic of this tissue; namely, dark lines standing out prominently (Fig. 44). They are the expression of the never absent overlapping, and the entire preparation resembles a veil which is partially folded. Admixtures of every kind, detritus, bacteria, remains of food, above all certain cells, are found almost invariably in the mucus. Only when the mucus is very glassy are these absent; their amount is increased the more turbid the mucus and the whiter its color. For the most part they consist of so-called disintegrated intestinal epithelia, i. e., homogeneous cells the component parts of which can no longer be recognized. This loss of structure Schmidt proved to be the result of imbi-

Fig. 43.—Mucus with isolated disintegrated epithelia. (Magnified 250 times.)

bition with soaps, for if the preparation is heated with acetic acid the cells become clear and the nuclei visible. The demonstration of half digested cells in the mucus is of significance. In such cases only cell nuclei may be found, and around these in cellular arrangement granules or crystals of bilirubin. The mucus itself is stained yellow by bilirubin. These signs certainly indicate that the mucus originates from the small intestine.

The fact that intestinal mucus contains epithelia is noteworthy when contrasted with the constituents of the mucus in sputum in which leukocytes predominate but only isolated epithelium is found. Under some circumstances, of course, pus cells also may be found in intestinal mucus. This is then of special import, a fact to which I shall revert later.

The diagnostic importance of mucus is based on the fact that every admixture of mucus to the feces, macroscopically and microscopically evident, denotes a pathological condition, this condition with but few exceptions being *catarrh* of the intestine. The meconium plug of the new-born has special significance, this being a short column of mucus which is expelled by the nursling even before meconium is excreted. This meconium plug was discovered only a few years ago by H. Cramer; that it escaped observation is probably due to the fact that it was covered in the diapers. Small mucus constituents are normally found in the feces of nurslings up to about the second week. It is still a question in how far the large mucus masses of colica mucosa which are usually passed without any fecal admixture, and which are regarded by Leube and Nothnagel as the result of a neurosis of secretion rather than an enteritis membranacea, may be referred to a mild catarrh.

Fig. 44.—The same after thorough admixture with dilute acetic acid. (Magnified 250 times.)

It may be said of the thin mucus coat which is occasionally observed upon the surface of hard fecal masses, and which resembles varnish after drying, that it is quite within the limits of physiologic conditions.

In the decision as to the *seat* of the catarrh, the question from which portion of the intestine the visible mucus present in the feces originates is of great diagnostic significance. We will observe that the excretion of mucus in the small intestine is less than that in the colon; furthermore that mucus is very readily digested, and when it is excreted in the small intestine it is subjected to the action of the proteolytic ferment, and that,

therefore, by far the greatest quantity of mucus particles admixed with feces originate in the colon.

When there is highly increased peristalsis of the small intestine and of the colon, mucus finds its way from the small intestine into the feces, which then, under circumstances already referred to, contain partly digested cells stained with bilirubin. This is chiefly found in enteric fever and in severe intestinal tuberculosis. Only the smallest, most finely distributed mucus flocculi can possibly originate in the small intestine; coarser particles undoubtedly come from the colon. A more definite localization in the course of this organ is only possible if we assume that the seat of the catarrh is the higher the finer the mucus flocculi, and the more intimate their admixture with feces. We may obtain some idea of the intensity of the inflammation from the number of admixed cells. As a rule, a white appearance of the mucus in consequence of the presence of considerable cylinder epithelium of the intestine much more certainly indicates catarrh than the presence of mucus which is glassy or but slightly turbid. Although the presence of mucus in the feces is conclusive, we dare not assume from the absence of mucus that catarrh is not present. Under some circumstances mucus may be found in the colon by the test washing of the bowel as advised by Boas.

Fig. 45.—Mucus with numerous red blood-corpuscles in the process of destruction; with these are crystals of triple phosphates. (Magnified 250 times.)

Small amounts of pure *blood* upon the surface of feces are found especially in the case of hemorrhoids, perhaps mixed with tough mucus. The muco-purulent dejecta in intestinal invagination are thin and profuse, and the so-called muco-purulent *lotio carnea* of dysentery also are often very profuse. The erythrocytes are readily destroyed in the intestinal mucus, hence they cannot be demonstrated by the microscope. In Fig. 45 we see red blood-corpuscles of a rusty appearance and embedded in the mucus in the process of destruction.

Altered blood coming from higher portions of the digestive tract is significant, but its demonstration is often difficult. Diagnoses based upon the macroscopic appearance alone are often erroneous, particularly when the feces are not of the typical tarry consistence but are compact.

Confusion with feces containing bismuth is precluded by the great number of crystals of bismuth suboxid revealed in every microscopical preparation. As a rule, only chemical or spectroscopic analysis will demonstrate the presence of hemoglobin.

In the recognition of ulcers or of malignant neoplasms of the stomach and intestine, paramount diagnostic importance has in the last few years been attached to the demonstration of minimal amounts of blood in the feces, the so-called *occult hemorrhages*. To obtain a positive result from blood tests, and this is especially true of the very sensitive Weber-van Deen test, it is necessary to exclude blood originating from food, therefore for several days prior to the investigation meat should not be taken by the patient; and we should also positively ascertain that small hemorrhages from the mouth or the pharynx are not the source of the bleeding.

TEST FOR OCCULT BLOOD

To carry out the Weber-van Deen test which is now popular, a large mass of feces (if the amount of fat is excessive perhaps after preceding treatment with ether) is mixed with water which contains about one-third its volume of glacial acetic acid until the mixture is of fluid consistence, and the hemoglobin is extracted in a test-tube by shaking with ether. To the ether which has been poured off ten drops of a freshly prepared guaiac tincture are added (one part of resin to 25 parts of absolute alcohol) and then (shaking after the addition of each drop) 20 to 30 drops are added of old turpentine oil which has been standing for several weeks in a flat bowl exposed to the air. A blue tint to the ether proves the presence of hematin. In shaking the ether with turpentine we should avoid covering the top of the test-tube with the thumb as the perspiration on the skin may affect the accuracy of the test. On account of the same fallacy in the test the presence of pus also must be excluded.

As to the well known hemin test of Teichmann, I think that, as a rule, too much table salt is used, a very small granule being sufficient; after heating we should wait a long time before examining the preparation so that the crystals may have time to form; finally, for unknown reasons the test is occasionally negative notwithstanding the positive presence of hemoglobin.

Pus cells in large quantities, partly with and partly without a mucus intermediary substance, are rarely found in the feces. Their appearance is, however, of great diagnostic importance and proves the presence of ulcers or a ruptured abscess. If merely isolated round cells permeate the fecal mucus this view is not permissible, and we can at most diagnosticate only an extreme irritation of the intestinal mucous membrane. The impossibility of demonstrating pus cells in the individual case by no means excludes the existence of ulceration, since small clumps in the diar-

rheic feces may escape recognition or they may have been dissolved by decomposition or the digestive ferments.

In exceptional cases *shreds of tissue* from the intestine, of ulcers, of hemorrhoidal nodules, or necrotic portions of the intestinal wall, especially in the case of invagination and of dysentery, may be desquamated and discovered in the feces. Their origin can be decided only by microscopic examination. They must not be confounded with tough mucus and animal membranes which are the residue of food.

This description of the investigation of the feces is by no means complete. Bacteriologic methods and the detection of animal parasites have not been touched upon. In the chemical aspect of the subject, clinical reasons constrained me to be brief; here much might be added. But it was my primary object to describe fully and especially emphasize the points most important to us as practising physicians, and clearly to portray the method of analysis of the feces which at the bedside will give us the most accurate information.

DIARRHEA, INTESTINAL CATARRH, AND INTESTINAL TUBERCULOSIS

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I. DIARRHEA

The designation *diarrhea* for those disturbances of intestinal digestion in which the discharges become more or less soft or fluid, and also are evacuated more than once during the day, originates from the view that food and drink are propelled very rapidly or almost rush through the intestine. This rapid propulsion through the intestine indicates *increased intestinal activity*; the appearance in the dejecta of constituents of the food points to *insufficient digestion and absorption*; the softer, more fluid composition of the discharges to *insufficient absorption of fluid,* and to *increased secretion* of the intestinal mucous membrane and the glands connected therewith. At all events, in diarrhea intestinal digestion is disturbed; therefore, all diarrhea is a symptom of intestinal dyspepsia.

As in gastric pathology we differentiate dyspepsias from organic gastric disease as purely functional disturbances, so we must discriminate between functional *intestinal dyspepsias* and organic diseases of the intestine. It would, however, be a great error invariably to characterize gastric or intestinal functional disturbances as nervous. Dyspepsia may, here and there, be of purely nervous origin; it is, however, the natural consequence of the condition that we do not sharply differentiate between intestinal dyspepsia and intestinal diseases, for sometimes intestinal dyspepsia is the precursor, sometimes an accompaniment, of an organic intestinal disease. Sometimes it exists independently, or the disease upon which intestinal dyspepsia is dependent has its seat in some other organ than the intestine.

Normally, the processes of digestion in the gastrointestinal canal are adapted to the ingested food. The function of the digestive glands is set in motion by chemoreflexes generated by the quantity and quality of the food and drink, and exactly regulated (Pawlow). The epithelia of the intestines facilitate the propulsion of the products of digestion, and their villi dip into the chyle, just as the roots of plants dip into the earth which furnishes their food, taking from it water, salts and other nutritive products which digestion has made fit for absorption, and which the organism requires for the structure of its tissues, for its growth, its development of force, the rehabilitation of what has been used, and for the

formation of heat. The unused remains of chyle, the residue of food, the products of excretion from the liver and the intestine itself, permeated by an enormous number of bacteria, are excreted by the intestine as feces.

The property by which the secreting and absorbing organs accommodate themselves to the nature and amount of the ingested food is, however, not absolute, but shows individual limitation. Therefore, a disproportion between force and amount may readily be brought about, and the juices of digestion and the power of absorption in the intestinal canal prove insufficient for the demands made upon them. The excess of chyle not taken up into the fluids of the body by digestion and absorption then abnormally irritates the small intestine, which attempts to rid itself of its irritating contents by increased peristalsis—diarrhea from the small intestine. In the less sensitive colon there may be compensation, and hypermotility, or diarrhea of the small intestine, may even be checked. True diarrhea, i. e., fecal discharges of soft or thin consistence, only occurs after decided irritation of the colon.

The stimulus for normal movements of the stomach and intestines originates from the contents, but in the intestines also from the chemical irritation of *products of fermentation and decomposition.*

To show the intensity of their effect upon intestinal movement, Bokai arranged the *organic (fermentative) acids* of the intestinal canal in the following order: Lactic acid, succinic acid, valerianic acid, butyric acid, formic acid, propionic acid, acetic acid, caproic acid and caprylic acid. The mildest irritation was produced by lactic acid, the severest by caprylic acid. These, as well as caproic acid, even in small doses produced in animals tonic intestinal spasm and decided hyperemia. Moreover, the foregoing acids not only caused diarrhea but, even in small amounts, produced catarrh; given in larger doses, decided inflammation of the intestinal tract. In contrast with these the salts of organic acids, even in much larger doses, produced no intestinal movements.

Of the *intestinal gases* hydrogen and nitrogen proved to be indifferent, while carbonic acid, marsh gas, and sulphureted hydrogen caused severe intestinal movements.

Among the *products of proteid decomposition,* Bokai found that *indol* had but slight action and *phenol* none at all, upon intestinal movements. On the other hand, even small doses of *skatol* produced severe peristalsis in the small intestine and rectum, and also gave rise to tonic spasm in these areas. Skatol at first causes the blood-vessels to contract, but in a few minutes this contraction is succeeded by a dilatation of the vessels. Skatol did not produce catarrh.

These reports indicate the great importance of intestinal bacteria upon normal and pathologic processes in the intestinal canal. It may, therefore, be interesting to discuss somewhat more minutely the *relation of bacteria to the intestine.*

INTESTINAL BACTERIA

At birth the intestinal canal contains no bacteria (Billroth), but each hour of extra-uterine life increases the number of bacteria conveyed into the intestinal canal of the nursling from its surroundings. As a rule, no harm is done the infantile organism by this bacterial invasion so long as the child is nourished by its mother's milk or that of a wet-nurse. The extraordinary sensitiveness of the intestine of the nursling to any other food, to cow's milk and to substitutes for milk, is, however, well known, and the marked tendency of infants to diarrhea, and the great mortality of children in the first years of life from gastric and intestinal disturbances, are usually attributed to the abnormal invasion of bacteria. Very interesting facts were communicated to us by v. Behring in his article "Diarrhea among Calves" (Kälbersterbe), and also concerning the mortality of nurslings in his well known publication "Säuglingsmilch und Säuglingssterblichkeit" (*Therapie der Gegenwart*, Heft I, 1904). In the dysentery of calves which is due to infection with the bacterium coli, and which may also develop in newborn calves by nourishing them with *boiled cow's milk*, according to Joest, the best prophylaxis has proven to be fresh milk from the mother cow immediately after the birth of the calf. Infant mortality can also be reduced from upwards of 30 to 3.55 per cent. by providing the child with a wet-nurse: Therefore, in breast milk, and in the milk of the mother animal, there exists a certain inherent property which protects the nursling and the sucking animal.

In the blood of cattle, v. Behring found isopathically developed antibodies for the most important intestinal bacteria, that is, for the bacterium lactis and bacterium coli. These protective bodies, as also the albumins and globulins of the blood and milk, enter the milk from the blood, and v. Behring, in fact, demonstrated that fresh cow's milk has a very active antibody antagonistic to the bacterium coli, and a weaker one to protect it from the bacterium lactis. The bactericidal property of milk is less than that of the serum, corresponding to the smaller percentage in blood albumin. These antibodies are destroyed by boiling the milk, but not by digestion; they may therefore pass through the intestinal wall of the nursling into its fluids in an unchanged condition. Upon the basis of these experiences v. Behring constructed his new process for the preservation of milk. The addition of formalin, 1 to 5,000 or 1 to 10,000, to the milk, as proposed by him, is said to increase the power of the antibodies.

With the advance of extra-uterine life, the organism itself produces certain protective products, the power of resistance in the intestinal wall increases, and, in consequence, the intestine of the nursling no longer acts like a filter with large pores, to which it has been compared. Verworn ascribes exclusively to the absorbent intestinal epithelia (which act like

ameboid cells) their discriminating faculty in the choice of food, inasmuch as, among all the constituents of the chyle, they take up only the fat globules, not bacteria, pigment granules, or other corporeal elements. As a matter of fact, intestinal bacteria cannot effect an entrance into the interior of the body so long as the intestinal mucous membrane remains normal and unimpaired.

The *number and distribution* of bacteria in the intestinal canal is not uniform. Billroth found the bacteria to be most scanty in the upper portions of the intestine; they constantly increase in number lower down, and are most numerous in the feces. With the rapid propulsion of the contents of the small intestine, the microbes from the stomach are comparatively soon carried into the colon; many of them fail to find favorable conditions for their development. For this reason the bacteria of the small intestine capable of sustaining life differ very markedly from those in the colon. The former are *almost wholly generators of fermentation,* and, while forming lactic acid, acetic acid, succinic acid and ethyl alcohol, produce decomposition, particularly in the carbohydrates of the food, while, under normal conditions, albumin in the small intestine is decomposed only in very small quantities by microbes.

On the other hand, proteid decomposition is paramount in the colon, hence, in this region, products of decomposition develop, such as indol, phenol, skatol, volatile fatty acids, aromatic acids, ammonia, carbonic acid, methane, sulphureted hydrogen and methyl mercaptan.

Macfadyen, Nencki and Sieber illustrate these facts by their report that in the cadaver of patients who were treated with bismuth the mucous membrane of the entire small intestine was only reddened, while the mucous membrane of the colon, from the ileo-cecal valve downward, had a black, velvety appearance in consequence of the reduction of bismuth by products of decomposition and the action of sulphureted hydrogen. A. Schmidt found the human small intestine, excised shortly after death and placed in a concentrated corrosive sublimate solution, to be of a greenish color; the mucous membrane of the colon, however, was red. The green color shows the reaction of corrosive sublimate upon bilirubin, the rose-red that upon hydrobilirubin, the product of reduction by the decomposition of bilirubin.

Fermentation and decomposition in the intestine do not exclude each other, although, under normal circumstances, their localization is different. If, under pathologic conditions, there is stasis of chyle in the small intestine, decomposition of proteids occurs there and leads to an increase of indican, to the presence of ethyl sulphuric acid ($C_2H_5HSO_4$) in the urine, and, in severe cases of occlusion of the small intestine (miserere), by regurgitation of the decomposed contents of the small intestine into the stomach, to fecal eructation and fecal vomiting. More frequent than decomposition in the small intestine is fermentation in the large intestine.

because in many forms of intestinal dyspepsia, starch-containing and cellulose-containing constituents of the food—particularly if insufficiently masticated—find their way into the colon, and there, by further fermentation, generate gases and acids.

We can scarcely form a conception as to the volume of bacteria present in the intestine. J. Strasburger has attempted to estimate their number in the feces by weighing, and found that, in round numbers, about *one-third* (32.4 per cent.) of the total solids of the feces in adults on an easily digested food consisted of bacteria. As the feces in the test diet employed by Strasburger (A. Schmidt's diet) contain upon the average 24 grams of total solids, we may say that a normal adult daily excretes about 8 grams of dry bacteria. The number of bacteria corresponding to these 8 grams Strasburger calculates at 128,000,000,000, while A. Klein by a previous computation found only about 9,000,000,000 (8,080,000,000).

On an attempt to inoculate plate cultures from definite portions of the feces with corresponding dilutions it was shown that the overwhelming majority of bacteria had already perished. Eberle could only develop upon gelatin 4 to 5 per cent., upon agar at blood temperature 10.6 per cent., of the bacteria found in feces. Among those capable of development the majority belonged to a *few constantly recurring* forms. On administering sterilized food, the saprophytes disappeared, until, finally, only the *bacterium lactis* and *bacterium coli* could be cultivated in the feces, therefore those bacteria against which the nursling is armed with protective products from the mother's milk.

What are the *bactericidal substances* which exert such immense and destructive power upon bacteria in the intestinal canal, even with other food than mother's milk? Nucleinic acid of the lymphoid cells (Kossel) possesses antiseptic and bactericidal properties, and, in another article in this volume, I have called attention to the importance of the tonsils and the lymphatic pharyngeal rings as protective organs. Although we have as yet no experimental proof, it cannot be denied that—apart from other functions—the extensive lymphatic apparatus of the intestinal wall, which is extraordinarily rich in follicles, not only acts as a filter in a physical sense but also chemically forms a protective process for the fluids of the body.

Antiseptic action must be admitted for the free hydrochloric acid of the gastric juice, although its importance is far less great than, for example, Bunge, Kast, and others have assumed. The acme of digestion, during which time free hydrochloric acid in the normal and healthy stomach possesses a concentration which permits antisepsis, is reached several times during the day; it lasts only for a few hours. In the remaining time this disinfecting action of hydrochloric acid is absent from the stomach. Hence numerous bacteria, without suffering particular damage, have an opportunity to invade the intestine from the stomach.

Finally, the biliary acids must be considered as antiseptic agents in the intestine. Maly and Emich report that the biliary acids set free from the cholates by the hydrochloric acid of the gastric juice, particularly taurocholic acid, have but little less of antiseptic action than salicylic acid and phenol. Naturally the disinfecting power of free biliary acids is only active while, and so far as, the chyle in the small intestine is of acid reaction. This period, however, is too brief to permit thorough disinfection.

If other bactericidal products should be found in the intestinal juices, we must assume that the varieties of intestinal bacteria which continue to live are those which, in the struggle for existence, feed upon the bacteria which constantly enter the gastrointestinal canal by means of the saliva or with food and drink. The different processes of this struggle are, at present, entirely beyond our knowledge. But the fact of its occurrence is proven by numerous experiments.

Thus R. Schütz, who introduced large quantities of Metschnikoff's vibrios directly into the duodenum of dogs through a fistula, was unable to cultivate this germ from the feces provided the intestinal activity of the animals experimented upon was normal and the intestinal mucous membrane uninjured. Bienstock fed garden earth containing tetanus germs to animals, but was unable to produce tetanus by inoculation experiments. The bacterium coli simultaneously cultivated with diphtheria bacilli in meat bouillon prevented the development of the latter, yet it succumbed to other microbes, for example, to streptococci and staphylococci.

Processes of fermentation and decomposition in the intestine are strongly influenced by the food, and the physiological and pathological processes in the intestinal canal and the changes in the fecal discharges are most intimately connected. These conditions may be readily investigated in small children.

The feces of healthy nurslings resulting from normal breast milk are voided two to five times daily, have an acid reaction, and an acid odor which is not unpleasant. They are of a yolk-yellow color, pappy, and according to the amount, the total solids form 1 to 1.3 per cent. of the dried food. Intestinal decomposition increases when nurslings are nourished with cow's milk; the feces are of lighter color, of firmer consistence, have an alkaline reaction, and the odor of feces or of decomposition. The amount of total solids rises to 2–3.1 per cent. of the dried food. Fermentation and decomposition in the intestine become more marked when children are nourished *ad libitum*. In such children the total solids of the feces rise to 5.9–7.5 per cent. of the dried food; therefore are five or six times as great as those of nurslings nourished with mother's milk (Biedert).

Infants who do not enjoy the blessing of mother's milk, and who are

"artificially" nourished, are greatly injured by over-feeding and overloading the stomach and intestines. If the child's stomach is not relieved by frequent vomiting, and the warning given by profuse evacuations of feces permeated with cheesy clumps and of disagreeable odor is disregarded, severe intestinal disturbances soon appear. These cheesy masses readily irritate the delicate intestinal wall mechanically, producing more violent peristalsis and the premature diarrheal evacuation of constituents of the food not yet absorbed and utilized. Added to this loss of nutrition, however, is the baneful factor that undigested, superfluous masses—the *deleterious residue of food* described by Biedert—promote a dangerous increase of bacteria in the intestine by furnishing a suitable culture media for the generators of fermentation and decomposition already present there, for the saprophytes which enter with the food, and, under some circumstances, for pathogenic bacteria. According to the nature of the food, a surplus of products of fermentation then produces *acid diarrhea,* or an abnormal proteid decomposition, *fetid diarrhea.* Other, often severe, forms of diarrhea which threaten the life of young children by intoxication or infection are produced by definite, more or less specific kinds of bacteria, and are especially influenced by season and climate, particularly by heat.

Adults who have no accurate knowledge or clear conception of the requirements of the body, and who, either from a love of eating or from habit, overtax their digestive organs by over-eating or by flooding them with wine and beer, and who thus fill the lower portions of the intestines with fermentative and decomposing material, suffer, as do young children, from "deleterious residues of food" in the intestine.

For a time the habit of over-distending the normal digestive organs produces no symptoms other than profuse and frequent compact or soft dejecta. This condition, designated by earlier authors as "*copropoesis,*" i. e., increased formation of feces, polycopria, is readily combined with a tendency to diarrhea, which, sooner or later, passes into habitual diarrhea that may continue for years and decades, often without much effect upon the general nutrition of the patient. This significant "*copropoesis*" and the tendency to diarrhea, if disregarded, are frequently followed in the course of time by severe injury to the intestine and other organs; for example, the liver, the kidneys, the heart and the vessels, and this is enhanced by advancing age and by certain occupations, for example, those necessitating a sedentary life. To the diarrhea which assumes a chronic form, flatulency is added, and the inflation of the abdomen, borborygmi in the intestines, and the frequent discharge of gas of a very disagreeable odor, become most annoying. The constantly distended intestine, especially the colon, gradually becomes blunted to irritation always present and loses its tone. The first sign of flaccidity—which precedes long torpor of the intestine—is bloating of the abdomen and the over-distention of

individual portions of the intestine or the entire colon from gas (partial or total *intestinal atony*). This flaccidity is not necessarily due to advancing age, for it may occur at any period of life, and an abdomen enormously distended with gas is especially conspicuous among poorly nourished children who suffer from chronic diarrhea.

Heavy food containing little of nutritive products rich in residue, especially of a vegetable nature, also causes overloading and extreme irritation of the intestine. We cannot nourish ourselves with just enough pure food according to its calory value, but require for a sense of satiety, and especially to stimulate peristalsis, the aid of food containing residue. If, however, the fluids of digestion are insufficient to assimilate and absorb a large amount of nourishment which, however, is poor in nutritive products, the undigested residue of the food will cause irritation, and the products of bacterial decomposition will give rise to diarrheic discharges which in the course of time are followed by catarrh and flaccidity.

The habitual overloading of the intestine and the consequent chronic intestinal dyspepsia which runs its course with diarrhea are opposed by an *occasional* form of acute intestinal dyspepsia due to *quantitative* errors in diet. Any excess in eating or drinking may bring this about. More or less severe intestinal disturbances which may also appear without pronounced symptoms on the part of the stomach are relieved in a comparatively short time if the intestinal canal becomes empty by the free discharge of gas and diarrheal evacuations. Without any excess whatever, a *qualitative* error in diet may be made unconsciously, or may pass unnoticed, and the error in nutrition may be recognized only by its consequences. Here individual circumstances, personal susceptibility of the intestine or the peculiarities of its bacterial flora play an important rôle. Some intestines are so irritable that diarrhea ensues on the slightest change in the food, and simple, non-irritating foods, which others always include in the dietary, act like purgatives. I do not refer to fresh fruits, vegetables, cucumbers, sauerkraut and fermenting drinks, which are known to possess laxative properties, but call attention to milk, for which some persons have an actual idiosyncrasy, and to which adults are often more susceptible than nurslings.

But the assumption of an abnormal or nervous *irritability of the intestine* will not explain all of the many acute and chronic forms of diarrhea, particularly when the so-called dyspeptic intestinal phenomena appear without an actual error in diet. After the ingestion of food hygienically suitable, and when the stomach functions normally, insufficiently digested chyle may be washed into the intestine by fluids, such as plain water, carbonated water, tea or coffee, taken while eating or soon afterward, and intestinal dyspepsia, abnormal fermentation, the generation of gas and diarrhea are frequently the consequences.

Exceedingly often the irritability of the intestine which, even with

ordinary food, causes intestinal dyspepsia and diarrhea, is the consequence of buccal dyspepsia or gastric dyspepsia. A disturbance of digestion in the mouth is due to insufficient mastication and to the incomplete admixture of saliva with the bolus, and occurs in all the affections of the mouth, particularly in caries of the teeth, with deficient dental prosthesis, and, much more frequently, from the bad habit of bolting the food. Even an excellent stomach is incapable of disintegrating large portions of food, nor can the intestine accomplish this, hence it suffers from the mechanical irritation of the fermenting and decomposing foreign body.

The various *gastric dyspepsias* produce intestinal dyspepsia and diarrhea in many ways. *Hypermotility of the stomach,* even when the secretory function is normal, fills the intestine to repletion with insufficiently prepared chyle. When there is a disturbance of gastric juice secretion, or a decrease or absence of hydrochloric acid, as in *achylia* or *apepsia,* the chyle also passes from the stomach into the intestine insufficiently prepared. If, in consequence of atonic conditions of the stomach—either with a normal or pathological secretion of gastric juice—hyperacidity develops from abnormal fermentation, even the upper small intestine simultaneously contains bacteria and large quantities of those acid products of fermentation whose stimulating effect upon peristalsis has been experimentally proven by Bokai. To all this must be added that, with the absence of hydrochloric acid in the gastric juice, the specific generator of pancreatic juice secretion is absent, and this renders intestinal digestion more difficult. In the undigested portions of the chyle excessive fermentation and decomposition take place. After what has been said, the mouth and the stomach may be quite properly designated as protective organs for the intestine. But all intestines do not require this protection, for frequently intestinal digestion is normal, and the state of the nutrition is good; often even constipation is present, notwithstanding these functional disturbances in the mouth and stomach.

Just as in the intestinal dyspepsias which have so far been described, in which products of fermentation and decomposition of the intestinal bacteria produce diarrhea by chemical irritation of the walls of the intestine, so this is also brought about by the products of metabolism of many pathogenic microörganisms.

The greatest irritation to the intestinal wall is caused by the *bacterial toxins* which are recognized as strong poisons. Toxic diarrhea in consequence of poisoning from meat, sausage, fish, mushrooms, potatoes, beans, etc., is more rarely observed than the sporadic forms, those occurring in families or in entire districts, the so-called diseases of the masses. The poisons are of *ectogenous* origin, and find their way into the body with tainted food, for *alkaloids of decomposition* and *cadaveral alkaloids,* so-called ptomains, do not develop under normal conditions of intestinal decomposition in the body. Brieger has found numerous products of this

kind in decomposing masses of meat. He observed that some of the bases isolated during the first period of decomposition (monamins and diamins) were physiologically indifferent, and some slightly toxic. After the third day of decomposition, however, more poisonous ptomains or toxins appear, and, on prolonged decomposition, substances of extraordinary toxicity (mydalein, methyl-guanidin and others). Even under pathological conditions, ptomain formation very rarely occurs in the intestinal canal itself, but invariably in *cholera,* and in a peculiar anomaly of metabolism, cystinuria (Neumeister). In pure cultures of the cholera bacillus and of the Finkler-Prior cholerine bacillus, after twenty-four hours, Brieger found pentamethylendiamin, and in older cholera cultures putrescin, cadaverin and methyl-guanidin. Therefore, intestinal autointoxication cannot be so greatly distributed nor so important as Bouchard maintains, although we refer to abnormal processes of decomposition in the gastrointestinal canal many general symptoms of intestinal dyspeptic disturbance, for example, lassitude and depression, headache, nausea and vertigo.

ETIOLOGY

Chemical substances and poisons introduced into the intestine from without irritate the intestinal wall just as do products derived from bacteria, and, by increasing peristalsis and the flow of the intestinal juices (secretion of dilution), produce more or less distressing diarrhea, hyperemia and inflammation.

Most of the milder *laxatives* act only by stimulating peristalsis, especially peristalsis of the colon; therefore, they free only the colon from feces and, at most, give rise to fecal diarrhea. The thin, fluid composition of the dejecta is due to its rapid propulsion through the intestine, and consequently there is diminished absorption of water. If we desire to induce increased secretion of the intestinal juices, or transudation from the blood into the intestine and watery diarrhea, alkaline salts are administered in *hypertonic* solutions. In spite of the existing osmotic differences in pressure, the current of diffusion from the intestine is only in the direction of the vascular tract and not in the contrary direction, because the normal intestinal wall is impermeable to fluids of the body (O. Cohnheim). Both properties, namely, the power of absorbing the intestinal contents and the impermeability of the intestinal wall to the fluids of the body, may be destroyed by poisons such as sodium fluorid and arsenic, or by either one of these. Very concentrated solutions of salt and sugar also injure the epithelium of the intestine so that the processes of diffusion follow only physical laws, for instance, fluid flows into the intestine. Thus the intestinal wall is irritated or the epithelium of the intestine is damaged by various spices, drastic purgatives, acids and alkalies, metallic salts and metalloids, alcohol, volatile ethereal oils, some vegetable alkaloids and

numerous chemical preparations which cannot here be individually enumerated.

Some of the substances mentioned may also produce diarrhea *by way of the blood,* for example, cathartic acid injected subcutaneously, colocynthin, aloin and others. Mercury, the mercurous chlorid of which is designated mite (hydrarg. chlor. mite), has a more highly toxic action from the blood than calomel from the intestine. Severe intestinal disturbance with superficial epithelial destruction or deeply invading necrosis of the mucous membrane has not rarely been observed from the external use of corrosive sublimate, for example, after vaginal and uterine douches. I have also known chronic diarrhea and colitis to develop in surgeons from the frequent disinfection of their hands with solutions of corrosive sublimate, and to be kept up by this method. The laxative effect of tobacco smoking appears to be due to the fact that nicotin is absorbed into the circulation from the oral cavity or the pharynx, or it may be absorbed by the lungs with the inspired air. Sometimes, however, the effect from tobacco is produced so rapidly—while at other times it does not appear at all in habitual smokers—that we must presuppose a suggestive action.

The diarrheas which occur at the onset or in the course of many infectious diseases are due to the circulation, the intestine showing no pathological change. In this category we include the loose stools observed in septicemia, in pneumonia, in malaria, in influenza and erysipelas, as well as those which take place in cholera and from putrid poisoning. The diarrhea which occurs in nephritis and uremia is the result of urea excreted from the blood into the intestine; in its transformation into ammonium carbonate this is very irritating, and even produces corrosion and necrosis. As a rule, we refer to *nervous influences* those forms of diarrhea in which no dietetic errors or pathologic conditions in the intestine can be recognized. Under normal conditions the digestive processes are controlled only by the gastrointestinal nerves; but these communicate with the pneumogastric nerve as well as with the sympathetic, with the cerebral and peripheral nervous systems; therefore, by these avenues abnormal processes and pathological conditions may derange the intestinal function. These functional disturbances may be motor, secretory, or sensory; they may continue for a long or short time, may recur periodically or become chronic, and may then produce clearly defined clinical pictures, for example, chronic diarrhea and peristaltic unrest of the intestine. In this manner, in the stomach as well as in the intestine, the various functions individually or collectively are abnormally *stimulated* or *inhibited.* This intestinal action may show variations, one function may be increased, another, on the contrary, may be inhibited or even absolutely arrested.

We are most familiar with the effect of sudden and great psychical stimulation upon the motor function of the gastrointestinal canal, and such disturbances in function are attributed to psychical irritation of the nerve

tracts which increases peristalsis or to psychical paralysis of the nerves which inhibit intestinal motion. Not infrequently, by *fright, anxiety,* or *fear,* the contents of the stomach are thus propelled into the intestine more rapidly than usual, and the intestines are more rapidly evacuated.

As to the effect of these involuntary and irresistible intestinal discharges, the laity in Germany have improvised characteristic designations for nervous people and cowards. Innumerable examples of this might be quoted from daily life; I shall limit myself to calling attention only to the increased peristalsis, the desire to evacuate the bowels, and even the diarrheic discharges (which actually take place even with repetitions) from which many persons of both sexes suffer during the excitement of a journey, while in company, before an appointment, preliminary to making a speech, in anticipation of going to court, etc. An auto-suggestive effect is produced by the mere sensation of traveling in a railroad car without toilet facilities, of being in a house or in company, etc., where it may be inconvenient to attend to the bowels.

Diarrhea due to the irritation of the skin by *cold,* and produced *reflexly,* is nervous diarrhea only in a certain sense, for example, when it follows chilling of the feet, a wetting, sitting upon a cold seat, or taking a cold bath; it may also arise from the diseased condition of organs adjacent to the intestine, especially the internal genitalia of women, and in genito-urinary diseases of men. During menstruation diarrhea dependent upon affections of the female genitalia shows periodic aggravation. There are also diarrheas which depend upon organic diseases of the spinal cord, such as the intestinal crises with diarrhea which periodically appear or periodically show aggravation.

In actual, chronic, uninterrupted, continuous diarrhea, varying only in intensity, I advise great deliberation before making a diagnosis of nervous diarrhea. I do not deny the occurrence of chronic diarrhea having a nervous or psychopathic foundation, but I believe it to be very rare. In many cases of this kind, if on strict diet, clinical observation and the regular examination of the feces will reveal the starting point of the irritative condition somewhere in the intestine, or external to it. The sequels of a former perityphlitis, a pericholecystitis, or other local inflammation of the peritoneum, must here be borne in mind, as well as also tuberculous ulcers in the rectum, polypi situated higher, frequently in women old and neglected lacerations of the perineum or prolapse, and in children enterozoa.

Whether the very persistent and exhausting diarrheas in Graves' disease, like the tachycardia, depend upon nervous hypermotility of the gastro-intestinal canal or whether they are due to autointoxication cannot with our present knowledge be positively decided.

SYMPTOMS

As a rule, the symptoms in diarrhea are quite uniform; those which have preceded or may follow the diarrheal discharges may show many variations, and those observed in nervous persons may bear a very peculiar stamp.

In some cases the patients complain only that the intestinal discharges are more frequent than usual, that their consistence is abnormally soft, or more or less fluid. Not rarely the preceding intestinal dyspepsia produces symptoms. A short time after the usual meal, often even during the meal, a distention of the abdomen is noted and is exceedingly unpleasant. This is followed by noticeable motion in the intestine, often only in the form of peristaltic unrest, such as gurgling and rumbling, but also often as increased peristalsis accompanied by slight pain. Other painful sensations may now and then be felt in the abdomen. These are caused by irritating food substances or abnormal digestive products, and indicate either local irritation of the mucous membrane—cutting pains, griping pains—or spasmodic contractions of the muscular layer of the intestine occurring in portions of the intestine already irritated, or they are due to the distention of isolated intestinal coils by gas or feces. Both spastic contraction of the intestine and great distention of the same—the former being a muscular pain, the latter a peritoneal pain—produce "belly-ache" or "griping," and when this becomes intense it is designated "colic." The paroxysmal, periodic appearance of these pains is characteristic; slight at first they increase in severity and become most intense; the cessation or disappearance of the pain, which resembles "labor pain" and is often compared with it, is also typical. In the intervals between the attacks the pain may cease and the patient seem restored to complete health; sometimes, however, in these intervals between the marked colicky attacks, a greater or less degree of sensitiveness remains until the irritating intestinal contents have been completely evacuated.

According to their cause, these colics have been designated *wind colic* (*colica flatulenta*) or *fecal colic* (*colica stercoralis*). Usually, however, these forms are combined. In these colics, the pain ameliorates as soon as gases or feces are discharged, the attacks are less frequent and the recurrences less severe. Inversely, however, they increase in intensity when the passage of gas or feces is prevented. The gases discharged may, according to their nature, produce a burning sensation which may continue for some time. This is also true of the feces, particularly the thin ones mixed with undigested fluids which corrode the anal mucous membrane.

These colics may be of such intensity as to cause even adults to writhe; in debilitated persons they sometimes cause syncope, and in children they frequently lead to general convulsions, even to loss of consciousness.

If we remember the physiology of intestinal movement, and particularly the fact that tonic contraction results from a local irritation of the intestine above the point of irritation, and that in consequence of this an annular constriction forms while the intestine below the point of irritation becomes flaccid and dilated, the frequency of invagination in small children, whose intestine, as is well known, is much more irritable than that of adults, is easily explained. As a matter of fact, during intestinal colic in children invagination of the small intestine and of the colon frequently occurs and is chiefly due to marked local irritation; often the colicky pains are the consequence of such invaginations. Spontaneous recovery no doubt takes place provided such invagination is but slight; in other cases intussusception of greater or less extent supervenes, and by constriction or occlusion of the intestine may threaten life.

Aside from complications on the part of the stomach (the intolerance of which toward any food or drink may cause debility, as in cholera nostras) the effect of diarrhea upon the general strength and nutrition depends chiefly upon the number and size of the evacuations during the day. The discharge of deleterious masses from the gastrointestinal canal may be beneficial, but since not these alone are discharged but also a quantity of nutritive substances, and especially much of the fluid of the body, all profuse diarrheas mean a loss of fluid to the organism. In children and debilitated adults, therefore, diarrheas produce a rapid loss of strength because such persons possess but little reserve material. After profuse diarrhea the turgescence of the skin rapidly disappears; the skin becomes flaccid and dry, the face hollow, the nose pointed, and the eyes are sunken in their cavities. Corresponding with the loss in water through the intestine, the amount of urine decreases, and the blood-pressure in some cases is so much reduced that the pulse becomes imperceptible. In severe cases in adults who evacuated from two to three liters of thin fluid feces in twenty-four hours, I have seen complete anuria which only disappeared when repeated subcutaneous infusions of normal salt solution were given or when the extreme thirst led the patients to supply the fluid loss by drinking. As a rule, a *restitutio ad integrum* rapidly occurs provided the diarrhea is not too long continued and is not due to severe anatomical lesions.

Nevertheless, even in acute diarrhea, the loss of fluid with the numerous, watery discharges is more or less injurious. The feeling of exhaustion, the cardiac asthenia in diarrhea, has been referred to the lowered blood-pressure from loss of fluid, also cramps in the calves and the appearance of tonic spasms in unilateral and symmetrical muscles of the extremities, of the trunk, and of the face, also in the muscles of the pharynx, larynx and eyes; in fact, the entire picture of tetany, including all its other symptoms, has been referred to inspissation of the muscular and nervous tissue from losses of water (Kussmaul, Fleiner). Other authors

attribute these severe museulo-nervous disturbances to gastrointestinal processes of intoxication. Their etiology, however, has not yet been decided, as no similar toxie products which have this effect have yet been proven to exist in the gastrointestinal canal of patients with diarrhea.

In most cases of severe diarrhea there is a loss of weight even if the condition persists but a few days. This, however, is rapidly compensated if no severe organic disturbanees are present, beeause the loss of weight is ehiefly dependent upon the loss of water. In prolonged or chronic diarrhea, the condition is different. Here, in the course of time, added to the loss in water is the absenee of resorption, therefore an insufficient ingestion of food, and, in consequence of this, a utilization of the reserve fund of fat, glyeogen and albumin of the body, therefore, under-nutrition and emaciation. In children and adults the latter may be of alarming extent; moreover, during this period of under-nutrition, in children the general growth of the body and also of individual organs eases, particularly that of the teeth and the bones, and a predisposition to other maladies, especially to infectious diseases, rickets, scrofulosis and tuberculosis, is established. Besides extreme emaciation, adults with severe forms of chronic diarrhea often show a peeuliar form of glossitis, apparently dependent upon atrophy of the epithelium at the tip and borders of the anterior half of the tongue, and atrophy of the papilla upon the dorsum of the tongue. The surfaee of the tongue is free from coating, it is smooth and glistening like the mucous membrane of the mouth, and very sensitive to ehemical and mechanical irritation. Small children, and adults greatly weakened by disease, partieularly the senile, not rarely succumb to profuse diarrhea. In eonsequence of the great loss of fluid and the excessive limitation of absorption, chronie diarrhea sometimes leads to severe forms of anemia, to cardiae weakness the result of fatty degeneration of the heart, to hydremia, and oeeasionally to albuminuria which finally causes death.

Middle-aged men who eat heartily and hastily, and who are aeeustomed to drink a great deal, withstand chronic diarrhea remarkably well for years and even decades. In spite of three, or many more, soft, diarrheie evaeuations daily, their general nutrition does not suffer; on the contrary, they inerease rather than lose in weight, and they are even indifferent to a decided degree of flatulence, until, from atony of the intestine, eonstipation, flatulent and feeal eolie, colitis or severe complications on the part of other organs (for example, the heart or the kidneys) and atheromatosis, ete., are superadded.

The reaction of intestinal disturbances upon the *nervous system* varies in its manifestations; as from the stomach so from the intestine every abnormal irritation may be conducted to the eentral organs of the nervous system by centripetal tracts in the pneumogastric and sympathetic nerves. In persons of neuropathic tendency, or those who are debilitated and

anemic, there are intestinal neuroses of the pneumogastric nerve which, similar to the gastric form, disturb cardiac activity and the respiration, and produce sensations of constriction, a feeling of anxiety, and vertigo. This so-called intestinal vertigo, which, as a rule, appears suddenly during a diarrhea of brief duration, and is sometimes synchronous with the fecal movements, may come in severe attacks or in a milder, but more protracted form; the latter is more likely to be induced by constipation than by diarrhea. There are, however, cases of paradoxical constipation notwithstanding profuse, even diarrheal discharges, and these conditions are frequently associated with such attacks of vertigo. In connection with intestinal dyspeptic disturbances, the pulse invariably becomes irregular and intermittent. To this peristaltic unrest of the intestine, which may be purely nervous or psychical but is usually due to abnormal intestinal irritation, general nervous excitement due to intestinal action is added, as well as noises of a gurgling character which at night prevent sleep, or cause the patient to awaken in a state of nervous anxiety which sometimes takes the form of definite fears (worms, frogs, etc., in the abdomen). Closely allied to these terrors is the hypochondriac depression, originating in sensations produced by peripheral irritation of the sympathetic, and referred by the consciousness to fear, which constantly centers attention on those organs and parts of the body which are threatened. Such individuals are actually tormented by their sensations and imaginations; they, however, make all those about them miserable, not less so their physician, or, more correctly, their physicians, who are annoyed much more than is necessary by their patients with intestinal hypochondriasis.

Migraine and hemicrania intestinalis, or hemicrania dyspeptica, are often produced by diarrhea; sometimes, however, migraine ceases with the appearance of diarrhea. The conditions are similar with hyperesthesia and neuralgia of other localizations, as well as vasomotor disturbances which, in a certain sense, are related to intestinal dyspepsia, such as urticaria, acute and chronic erythema, and acne-like eruptions of the face, whether this relation be established by the nerve tracts or by the circulation, by reflex action and irradiation or by intoxication.

DIAGNOSIS

The diagnosis of an intestinal affection must not be based upon the recognition of one particular symptom of the disease—for example, diarrhea constipation, colic, or hemorrhage—but a number of problems must be taken into consideration. In diarrhea, for instance, the cause and the seat of the disturbance must be determined, and we must decide whether a merely functional disturbance—hypermotility, hypersecretion, or diminished absorption—is the basis of the malady, whether this functional disturbance is the expression of pathological changes in the intestinal wall, and whether

these are of substantive character; therefore, whether they are of intestinal origin, are dependent upon disturbances or diseases of other organs, or are due to general constitutional conditions. If the latter, the organs directly connected with the intestine must, above all, be investigated; first the oral cavity and the stomach, then the liver and the pancreas, later the peritoneum and the organs related to it, and especially the female genitalia, then the nervous system, the composition of the blood, the organs of circulation, and, finally, the kidneys. The clinical examination of the entire body and its individual organs is absolutely necessary to determine the origin and the action of processes of disease in the intestines. Of these we actually get but imperfect knowledge, and this chiefly by an *examination of the feces,* which occupies the same position in the diagnosis of intestinal diseases as the examination of the gastric contents in the diagnosis of gastric diseases, the examination of the sputa in the diagnosis of respiratory diseases, or the examination of the urine in renal diseases and in disturbances of metabolism.

Examination of the gastric contents and the intestinal discharges has not as yet attained such popularity in practice as examinations of the urine and the sputum. The methods are more complicated and difficult, nevertheless the pith of a voluminous literature of investigations of the gastric contents and their value in practice is infinitesimally small and simple. Contrasted with a pure, scientific *coprology,* those methods of examination of the feces which permit a positive clinical diagnosis and furnish clear therapeutic indications are just as simple. First among these methods is *coproscopy,* simple inspection of the feces. When the unaided eye is not sufficient the lens and the microscope should be employed, or even chemical reagents. Quantitative analysis of the feces is only rarely necessary in practice, although its value in solving scientific and physiologic questions in the laboratory cannot be overestimated.

Like everything else, coproscopy, the inspection of the feces, requires practice, and, if we expect it to yield results, inspection must be not only occasional, but regular and continuous until the diagnosis becomes absolutely clear or the patient ceases to be under observation. In order to secure any reliable data from the examination of the feces, we must have a knowledge of the patient's previous diet. When the findings are doubtful, the question will arise whether other foods, drink, or drugs have been taken.

For example, let us consider a profuse, diarrheic stool in which large and small particles of solid food of various kinds may be recognized with the naked eye: This form of diarrhea has long been known as *lientery.* The investigation is made in order to decide *which constituents of the food have been carried or propelled rapidly through the intestine, and what abnormal products the intestine has contributed to the fecal admixture.* The size and amount of the undigested particles of food evacuated

lead us to suppose that an abnormally large amount of food was eaten, that it was hastily swallowed, i. e., was insufficiently masticated. Consequently, we examine the teeth of the patient, order a reduction in the amount of the food, and insist that in the preparation of the food in the kitchen it be more finely divided by hashing or other mechanical means, or that this be done during eating either by artificial or natural mastication (a masticator). Coproscopy, repeated the next day, may reveal a normal condition. Therefore, there was no actual intestinal disturbance in the previous lientery but the cause was merely a poor condition of the teeth, incomplete mastication, or overeating.

If in a given case, *notwithstanding a prescribed diet,* undigested particles of food appear in the feces, this variety of lientery admits of quite a different interpretation. We now investigate the character of these unabsorbed constituents of the food. If the fecal discharges are thin this is sometimes very easy, for the solid constituents may be extracted by the aid of a pipette or a small stick. It is sometimes advisable to receive the latter in a wire net or fecal sieve (Boas). Necessarily, compact feces must be carefully broken up in a vessel with water by a pestle.

The most valuable elements in our food are *albumin* (meat), *fat,* and *carbohydrates,* while the residue of vegetables is of subordinate importance. Hence, when we find with the naked eye or with the microscope fragments of food in the feces we know that we are dealing either with an albumin (or *meat*) *diarrhea* or a *fat diarrhea,* that is, *albumin,* or *meat,* or *connective tissue lientery* (*proteorrhea*) or a *fat lientery* (*steatorrhea*). Accordingly, with the relatively rare appearance of starch in the feces, we designate the affection as amylum lientery or amylorrhea (v. Oefele).

The results of the physiology of digestion teach us that albuminous products are digested in the stomach by pepsin and hydrochloric acid, and in the intestine by the proteolytic ferment of the pancreatic juice (trypsin), while fat, which is only slightly absorbed in the stomach (Volhard), is chiefly absorbed in the small intestine by the steapsin of the pancreatic juice. The carbonated alkali of the pancreatic juice, the bile, and the fluids of the small intestine combine with free fatty acids, and, by giving off carbonic acid, fatty alkalies or soaps develop at any stage between the molecules of neutral fat which have not as yet been split up. Thus a fine emulsion of microscopically small fat globules is formed in the intestine. The digestion of carbohydrates begins in the mouth by the action of the ptyalin of the saliva during mastication. If the stomach contents have an acid reaction, amylolysis ceases in the stomach; but it begins again in the small intestine by the action of the ptyalin of the pancreatic juice.

The most important carrier of albumin is meat. From what has been previously stated we know that the presence of undigested meat in the feces indicates a disturbance of gastric or pancreatic digestion in the small

intestine, and a careful investigation of the functions of the stomach in the usual manner is advisable. The result will show whether the disturbance is gastric or pancreatic, and whether we should prescribe hydrochloric acid and pepsin or a preparation of pancreas. To complete the technic of fecal examination, it must be mentioned that the passage of undigested meat is evidence of impaired gastric and pancreatic digestion. Connective tissue, according to Kühne, is digested in the stomach; *the finding of shreds of connective tissue in the feces therefore indicates a lack of gastric digestion,* HCl-anacidity, achylia gastrica, or apepsia. Muscle fibers are digested in the small intestine by trypsin; hence the macroscopic recognition of remains of muscle or well-retained transverse muscle striæ by the microscope indicates a *disturbance of digestion in the small intestine*—deficient trypsin digestion. The passage of undigested remains of milk in the form of cheesy flocculi is subject to the same interpretation.

With a little practice, an enormous increase of fat in the stool may be certainly recognized by the oily, creamy consistence, the pale, grayish, yellow or grayish white fatty gloss, by the fact that the feces float in water, and their behavior when pulverized (a membrane showing a play of colors). Under the microscope such feces show unsplit, neutral fats in globules, and fatty acids and soap needles in profusion. On analyzing these findings, we are forced to consider *disturbances in the flow of bile and in bile production*; therefore, diseases of the liver and biliary passages (occlusion of the common bile duct, jaundice), derangement of the pancreatic secretion, and actual diseases of the small intestine. Doubt can only be removed by clinical observation combined with therapeutic measures (pancreatic preparations) and chemical examinations of the urine and feces, which cannot be considered in this article.

The carbohydrates of the food, provided this is properly prepared in the kitchen and thoroughly masticated, are usually well digested. The discovery of food particles containing starch indicates, therefore, an extremely rapid propulsion through the intestine. Since, however, the carbohydrates which escape digestion and absorption ferment and rapidly decompose in the intestine, we are justified in concluding from the presence of large quantities of acid products of fermentation (acid reaction of the feces) or a great volume of gas in the bowel or in the feces (perhaps A. Schmidt's fermentation test) that there is deficient carbohydrate digestion in the intestine. The absence under the microscope of the iodin reaction is not always conclusive; digestion of starch may be deficient and the reaction for starch in the feces be very slight provided the undigested starch has been destroyed by fermentation.

We must search for the cause of poor digestion of starch in the improper preparation of the food, in imperfect mastication and insufficient admixture with the saliva, also within the bowel itself (intestinal fermentative dyspepsia).

The treatment of acute and chronic diarrhea will be considered in the chapters relating to intestinal catarrh.

II. INTESTINAL CATARRH

Diarrhea and intestinal catarrh running its course with diarrhea present in common the disturbances in function previously described. Often, therefore, they are confounded with one another. This error, however, is not so great as it may appear; for between diarrhea and intestinal catarrh there is often a difference only of degree, and simple diarrhea may precede true intestinal catarrh, may continue as a functional irritation, and cause a catarrhal inflammation of the intestinal mucous membrane. The chief difference between diarrhea and intestinal catarrh consists in a pathologic change in the intestinal wall which may be clinically recognized from the admixture of mucus to the diarrheic stools.

ACUTE INTESTINAL CATARRH

This is one of the most common maladies, and occurs in man at every age, but especially in small children, in the aged, and in the debilitated who have little power of resistance. Acute intestinal catarrh is usually a primary and substantive affection, more rarely it occurs secondarily and symptomatically in the course of previously existing diseases.

Etiology.—The most frequent cause of intestinal catarrh is the abnormal nature of the intestinal contents which immoderately irritate the intestinal mucous membrane, either mechanically or, more frequently, chemically. Those causal factors which have been enumerated for the development of diarrhea may also be of etiologic importance in intestinal catarrh. Among the normal products of fermentation and decomposition of the intestinal contents which are of great importance in peristalsis, we mention also those which, according to Bokai's investigations, not only produce diarrhea, but also spastic contraction of the muscles of the intestine and catarrh and inflammation of the gastric mucous membrane. The same dietetic indiscretions, the immoderate eating of food and drinking of fluids, or the unsuitable nature of these and their injurious effect upon the body —briefly, all errors in diet of either a quantitative or qualitative nature— may in the one case produce only diarrhea, and in another give rise to intense, catarrhal, intestinal symptoms. Local and periodic conditions which increase the virulence of bacteria may favor the ectogenous decomposition of food, so that the toxic action of the abnormal products of decomposition not only results in functional irritative symptoms but also in pathological changes in the intestinal wall. In the intestine itself a foreign vegetation introduced with spoiled food may replace the normal intestinal flora or even exceed this in growth, and by abnormal

decomposition also generate products in the intestinal contents—therefore endogenous—which damage the intestinal mucous membrane. This *chyme infection* of Escherich is the opposite of *intestinal infection*—the direct damage of the intestinal wall by bacteria and other specific pathogenic agents.

Many infectious diseases begin with the appearance of gastrointestinal catarrh, which, as in the case of enteric fever, cholera, and dysentery, may be attributed to the accumulation and development of specific pathogenic microörganisms and their effect on metabolism. In all probability, the majority of the pathogenic microörganisms are capable of producing infectious intestinal catarrh; this is certain, at least, of *staphylococci* and *streptococci*. Similar in action to bacteria are *amebæ, coccidia* and *infusoria*, which are common in the tropics and find entrance to the intestine by means of stagnant or decomposed water. Upon entering the mucous membrane they cause the death of the epithelium by a poison that they develop, and even in the deeper layers of the intestinal wall they produce inflammation and necrosis which, in the severer forms of enteritis, lead to ulceration. Just as abscesses develop in the liver by means of the portal vein circulation in dysentery and in some forms of ameba enteritis, so may metastatic inflammation and septicemia follow infectious intestinal catarrh. The conditions are the reverse in other infectious diseases which are not localized in the intestine; for example, in puerperal fever and other septic processes, also in the *dyscrasias* which are the consequence of some infectious diseases (tuberculosis, syphilis, malaria and pyemia, as well as in carcinomatosis and in uremia) by producing an abnormal composition of the blood or, on the contrary, the excretion of definite toxic-acting products into the intestinal canal, which there produce intestinal catarrh.

In the forms of disease last mentioned, the mucous membrane of the entire digestive tract may be the seat of catarrhal inflammation, for in catarrh due to abnormal intestinal contents the localization and the distribution of the catarrh depend upon the extent of the *materia peccans* into the intestine, the length of time it retains its noxious properties, or where it assumes this virulent nature.

An acute gastric catarrh distributes itself within the upper small intestine, not, as was formerly assumed, by the extension of the inflammatory process from the gastric mucous membrane to the duodenal mucous membrane, but by the propagation of the same irritating, deleterious agents. The lower portions of the intestine may be exempt when chyle, which is still abnormal in the duodenum and in the jejunum, becomes innocuous by further dilution with the digestive juices, or by dilution with a large amount of fluid which has transuded into the intestine, or when it is coated with mucus. On the other hand, abnormal processes of decomposition may frequently be generated in the deeper portions of the small intestine or in

the beginning of the colon, in regions where the conditions are more favorable for the existence of microbes than in the upper portion of the small intestine or in the stomach.

Except when caused by abnormal fermentation and decomposition, catarrh of the colon is most frequently caused by the abnormal dryness and hardness of the feces, by their prolonged retention, or by their accumulation in large masses. Owing to peculiar local and functional conditions, definite areas of the colon are predisposed to catarrhal and inflammatory affections; above all, the cecum, the flexures at either side of the abdomen, and the sigmoid flexure. The same is true of the rectum; but, besides accumulated feces in the ampulla, various kinds of ingesta produce catarrh of the mucous membrane, as do also *enemata* which irritate thermically, *nutritive enemata* which irritate by their concentration or by the decomposition of food products which they contain, and *drugs* which are injected into the rectum. Occasionally gonorrheal virus produces a severe catarrh of the rectum. Refrigeration must frequently be considered as a cause of acute intestinal catarrh, and also, finally, intestinal parasites and foreign bodies which find their way into the intestine.

Secondarily or symptomatically intestinal catarrh arises in other pathological, structural changes of the intestinal wall, in many ulcers, in inflammation of the peritoneum, and in local and general circulatory disturbances which affect the intestinal canal. Abnormal displacements and the fixation of individual intestinal coils, also invagination, torsion, and incarceration, lead to stretching and compression of the mesenteric vessels, and, consequently, to local circulatory disturbances, changes in the mucous membrane, and intestinal necrosis. In all of these conditions it is true that intestinal catarrh, as such, is of minor importance.

Pathology.—In acute intestinal catarrh the pathological lesions are often out of proportion to the severity of the clinical symptoms, especially in the cases which run a rapid course and in which severe intestinal infections or intoxications have caused a speedy death. In these instances, chemical and bacteriological investigations lead to more accurate conclusions than the autopsy findings.

When a catarrhal enteritis is present the intestinal tract only exceptionally shows inflammatory change; as a rule, merely isolated portions are diseased, some more, others less, while extensive areas of the intestinal canal are often entirely exempt. The diseased mucous membrane may be covered with a slimy, or, more often, a serous, or, at other times, a mucopurulent and turbid exudate from desquamated epithelia and pus, and this may sometimes be of even a hemorrhagic nature. The mucous membrane itself is flaky or diffusely reddened and swollen, is more markedly infiltrated than normally, often showing hemorrhagic infiltration or permeation by small hemorrhages, particularly in the crests of the mucous membrane folds.

Many intestinal catarrhs are characterized by swelling and redness of the follicular apparatus (enteritis follicularis). The solitary follicles then form reddish or grayish white prominent nodules, and on the agminate glands (Peyer's patches) are grayish white or gray elevations surrounded by a hyperemic area, and permeated by groove-like depressions.

Microscopically, the mucous membrane is markedly hyperemic, and frequently shows a very defective epithelial coating as a post mortem phenomenon. The epithelia still present have undergone a mucoid and destructive change, the supporting tissue and the submucosa, according to the intensity of the inflammation, give evidence of serous transudation and small cell infiltration, while, on the other hand, the muscularis is slightly or not at all altered.

In accordance with the localization of the inflammation, we differentiate pathologically between duodenitis, jejunitis and ileitis, a typhlitis (appendicularis and typhlitis cecalis), colitis (even sigmoiditis) and proctitis. Clinically, these differentiations are impossible.

As a rule, in the milder forms of acute intestinal catarrh complete recovery occurs. After the removal from the intestinal canal of all deleterious products the inflammatory irritation ceases as well as the pathological secretions, the products of inflammation are absorbed, and the epithelium which has been destroyed is replaced by regenerative proliferation of the epithelial cells retained in Lieberkühn's crypts.

If the inflammatory irritation is long continued, or if it recurs rapidly and often, chronic intestinal catarrh may develop from an acute form. More intense irritation leads to severe inflammation of the mucous membrane and of the deep layers of the intestinal wall, to necrosis and to ulceration. Severe infectious and toxic forms of enteritis not rarely result in death, and they form the chief cause of the enormously great mortality of infants.

Symptoms.—Among the clinical symptoms of acute intestinal catarrh the most common are intestinal motor, sensory, and secretory irritative phenomena: *Diarrhea, pain* and the admixture of *mucus* to the feces. In the severe infectious and toxic forms of intestinal catarrh to these must be added the *general symptoms of infection and of intoxication* which not infrequently may be of very serious nature and extremely dangerous to debilitated persons.

Among the symptoms, diarrhea and the pain of various kinds so often combined with it have been described. I have also emphasized that an intense irritation of the colon is a typical characteristic of the diarrhea. In fact, there are many forms of catarrh of the small intestine which cannot be recognized except by those who make it a rule in all gastrointestinal affections to subject the feces to regular and minute examination. Duodenal catarrh, for example, may be masked by the symptoms of gastritis, with which it is usually associated until the appearance of jaundice, which

originates from the duodenum. Catarrh may also be of the jejunum and of the ileum; in fact, catarrh limited to the small intestine is frequently unrecognizable because it has so few diagnostic points of support. It is true that it may run its course with marked disturbance of the constitutional condition and local difficulties, but diarrhea is absent, or present for only a few days, when the colon also becomes irritated and the seat of catarrhal inflammation. Nothnagel has described as *jejunal diarrhea* a condition in which feces are evacuated which are of the same composition as the contents of the jejunum: Odorless masses with unaltered constituents of the food and unaltered bile, and permeated with mucus. Such diarrheas occasionally occur. Much more frequently, however, catarrh of the jejunum and catarrh of the upper ileum run their course without diarrhea, at least for a time. Not rarely we observe in such cases irritative symptoms which usually precede diarrhea—increased peristalsis, rolling movements, borborygmi and pain—but the diarrhea does not occur for some time. Notwithstanding this—probably in consequence of the too rapid propulsion through the small intestine of the undigested remains of food—abnormal fermentation generates in the large intestine an increased gas formation which causes some distress, and which may be clinically recognized until, after more or less time, diarrhea supervenes. In the evacuated feces insufficiently digested fragments of food will probably be found, but often, at the onset of diarrhea, no unaltered bile. Only in catarrh of the small intestine and colon combined, which runs its course with frequent diarrhea, is bile again passed in an unaltered condition. In such feces we sometimes find, besides unaltered bile, also other digestive juices, or, at least, their ferments, pepsin and trypsin. On exposure to the air their bilious tint occasionally changes and becomes greenish, or leek-colored.

Abdominal pain is common in most cases of intestinal catarrh. This is usually dull, sometimes of a burning or cutting character, and may be continuous or increase and decrease periodically. This may spread over the entire abdomen, or may be localized in the region of the navel, especially if the small intestine, in particular, is implicated. Acute catarrh of the colon appears to cause much greater pain than that of the small intestine; the pain is localized in the ileo-cecal region, in the flexures of the hypochondriac regions, and in the sigmoid flexure, or is often confined to the transverse colon or may extend throughout the entire course of the colon.

Pains of the greatest intensity may be caused by a simple *mucous membrane catarrh of the appendix*; very frequently this suggests perforation; however, peritoneal phenomena and fever do not follow, and if, in such cases, as a precaution, the appendix is resected, the slight change in the mucous membrane of the excised appendix is surprising. It may not be out of place to emphasize the fact that the vermiform appendix is

predisposed to extremely painful colics which appear upon the slightest irritation.

Diarrheal discharges from the large intestine also cause periodic attacks of colic which are frequently of great intensity. As already stated, these are attributable to spastic contracture of the annular musculature of the intestine or to the great distention of certain portions of the intestine by gas or feces (incarcerated gas, wind colic), while the non-spasmodic intestinal pains are to be ascribed to chemical irritation of the sensory nerves of the intestinal wall. In many intestinal catarrhs pain is absent; the diarrheic stools are forcibly ejected from the bowel.

Proctitis.—Acute proctitis is accompanied by peculiar pain which in mild cases shows itself by a more or less distressing sensation of burning, often by ineffectual attempts to evacuate the bowels, and, in severe cases, appears in the form of spasmodic tenesmus. Not infrequently—although but little fecal matter is discharged—the mucous membrane of the rectum to a greater or less extent protrudes from the anus as a tumor, or tumor-like mass, almost ectropic. Occasionally, as in colica spastica, spasm of the anal sphincter appears and may continue for some time, giving rise to severe pain, and preventing evacuation. Often in acute proctitis mucus alone is discharged, sometimes of a glassy or shreddy character, occasionally also in peculiar globular or cluster-like masses; when there is severe tenesmus, besides mucus blood also invariably appears in the dejecta. Often hemorrhagic mucus, or blood alone, is noted without ulcerations being necessarily present in the rectal mucous membrane.

In acute intestinal catarrh the general systemic disturbances vary extraordinarily. Robust persons who have no fever very often pass through an attack without being confined to bed; the evacuations, which often amount to ten or twenty during the day, are very distressing, as well as the feeling of soreness about the anus and the lassitude which is attributable to the great loss of fluid and the impossibility of taking sufficient nourishment, but, in a few days, the difficulty passes away. In severe cases there is a sensation of extreme illness which renders the patient incapable of attending to his work, and confines him to bed; so fulminant do the symptoms appear that a severe infectious disease or an intoxication is supposed to be present until the diagnosis becomes clear.

Fever is more commonly observed and is also higher in children than in adults; the onset and course, however, conform to no distinct type. In the toxic forms of acute intestinal catarrh, the temperature is apt to be subnormal.

Other symptoms frequently observed as accompaniments of dyspeptic disturbances, such as headache, lassitude, nausea, anorexia, coated tongue, thirst, an eruption of herpes about the lips, urticaria, pains in the limbs, also cramps in definite muscle groups, and cramp in the calves, are noted also in acute intestinal catarrh. In severe cases typical attacks of tetany

and delirium are now and then observed. In children the nervous disturbances of acute enteritis are particularly severe and, at the onset of the disease, tubercular meningitis may be simulated.

Corresponding in ratio with the losses of water by the copious bowel discharges, at the height of an attack of acute intestinal catarrh the excretion of urine is very deficient, and the daily amount voided is much reduced; often anuria may last for a day or even longer; during this time blood-pressure is decidedly lessened, the pulse is small and often irregular. With the reestablishment of diuresis, albumin may often be found in the urine as well as casts, both, however, only temporarily.

Upon examination the patients, particularly children, are found apathetic and apparently seriously ill. As a rule, very little can be demonstrated objectively except the frequent diarrheic discharges at the onset of the disease. Enlargement of the spleen has now and then been observed in infectious catarrh. The abdomen is usually unchanged, at times somewhat retracted, at other times distended. Through the thin abdominal walls we occasionally perceive the motions of the intestine, or they may be felt with a palpating hand, or rumbling is heard in the abdomen and portends another intestinal evacuation. As a rule, the entire abdomen is sensitive to pressure, but it may be so in only limited areas; gurgling is readily produced in the ileo-cecal region. Profuse accumulations of fluid and intestinal contents here and there cause dulness upon percussion. The comparatively rapid change in the symptoms will, however, soon show how erroneous is the assumption of a peritoneal exudate or an ascites.

In no other malady is the destructive action of the affection so distinctly apparent, and in relatively so brief a time, as in severe acute (gastro-)enteritis. In severe cases striking changes may take place between one visit and another. A blooming child may be changed to a pitiable object, the previously full, round face may be emaciated, a formerly strong heart may be flaccid and weak. Just as rapidly, however, a change for the better may supervene when the profuse discharges cease and copious draughts of fluid have quenched the thirst of the body and its tissues.

Course of the Disease.—In adults the course of acute intestinal catarrh is usually favorable, provided there is no severe alimentary intoxication. Unfortunately, at the onset of an acute enteritis we do not always know that it is the primary effect of an infectious disease—occasionally the latter is, in fact, the case, under which circumstances only in the further course of the disease do the characteristic symptoms of specific infection, influenza, articular rheumatism, enteric fever, or cholera, appear. If, however, there is no reason to suspect these affections, the prognosis in adults is always favorable. In a few days the affection, whose severity is usually limited to the excessive number of bowel evacuations daily, has run its course, and although convalescence may be somewhat pro-

traeted the duration of the disease is rarely more than one or two weeks. Only in little children is acute enteritis, particularly gastroenteritis, a severe disease which greatly influences the general mortality.

In persons of any age severe acute intestinal catarrh not rarely leaves an irritative condition of the colon which expresses itself by a tendeney to diarrhea, and may persist for a considerable time.

Diagnosis.—The diagnosis of acute intestinal catarrh is, as a rule, very easy; but the localization of the catarrh, and, in some cases which run their course with severe constitutional symptoms, the decision of the question, whether the disease is an infection or intoxication, may be difficult.

In practice, usually, no line of demarcation is drawn between diarrhea and intestinal catarrh, and, indeed, the designation intestinal catarrh appears to be the more popular one for the cases under eonsideration. The chief characteristic of catarrh, however, is the admixture of mucus with the feces, and this cannot be overlooked if the investigation of the feces is at all thorough.

We regard mucus as a pathological seeretion, the presence of which in a given case clearly indicates that the limits of a purely functional disturbance have been exceeded, and that pathological changes have taken place in the intestinal wall; namely, catarrh and inflammation. *The differential diagnosis between diarrhea and intestinal catarrh can, therefore, be made only by coproscopy.*

Mucus is a glycoproteid which upon splitting beeomes a protcid and a carbohydrate. This splitting may be produced chemically (by boiling with a 5 per cent. hydrochloric acid solution) after which the albumin body, as well as the carbohydrate, may be demonstrated by reagents. It takes plaee in the stomaeh as well as in the small intestine; therefore, *mucus is digestible.*

Conscquently sputum that has been swallowed, mucus from the pharynx, from the esophagus, or from the stomach never reappears in the feces; therefore, mueus found in the feces must originate in the intestine. But mucus is also digested in the upper portion of the small intestine; in the colon, however, it is certainly not. Hence it is possible that mueus originating from catarrh of the upper small intestine disappears wholly or entirely in digestion, provided it is not too rapidly propelled into the lower portion of the intestine and voided with profuse diarrheal discharges.

An admixture of mueus with the feces, like the more or less intimate admixture of blood, indicates a somewhat high point of origin. Floeculi of mucus intimately combined with feces, and bile-stained, denote catarrh of the small intestine. This sign is, however, not absolutely eertain, for in severe diarrhea unaltered bile may pass through the entire intestinal canal and be readily recognizable in the feces. In doubtful eases, Gmelin's test or Schmidt's corrosive sublimate test is decisive for unaltered biliary coloring matter. This reaction shows an abnormally rapid propulsion

through the intestine of all those constituents of the feces which are stained with unaltered biliary coloring matter (and which become green with Schmidt's test).

While a microscope is often necessary to detect small mucus flocculi (and partly digested ones?) from the upper small intestine, mucus from the lower small intestine, also that which comes from the colon, even when intimately admixed with feces, can always be recognized by the naked eye. If only the descending colon, the sigmoid flexure and the rectum are diseased, the mucus masses originating from these areas may easily be detached from the feces; they will rarely show staining with bile. In acute catarrh of the rectum and the descending colon pure mucus is sometimes discharged. An admixture of blood with the mucus or feces is very frequently found in acute proctitis running its course with severe tenesmus even when there is no ulcerative process in the rectum.

The quantity and composition of the mucus admixed with the feces varies greatly; between the finest flocculi, delicate membranes, and large shreddy membranes, all intermediate stages may be observed. Amorphous mucus gives to many diarrheic stools a gelatinous composition; sometimes amorphous mucus is discharged alone as a glassy mass. The uniform admixture of small threads of mucus with diarrheic stools gives to them a choppy appearance (thread-worms).

In the descending colon and rectum the mucus sometimes appears in the form of bands or tubes corresponding in shape with these portions of the intestine. A peculiar globular or grape-like appearance of the mucus, which to the inexperienced sometimes becomes a diagnostic enigma, is acquired in the haustra of the colon or the ampulla of the rectum (in chronic catarrh more frequently than in acute, especially after convalescence has set in) when the feces which are propelled by peristalsis toward the anus take from the mucous membrane its mucus coating and roll it up. Admixture of mucus with the feces does not take place in the lower portions of the intestine; the feces then are covered with mucus—actually enveloped in it.

Microscopic examination of the mucus will protect us from a possible confusion with the remains of food, and at the same time it reveals the intensity of the catarrhal inflammation; this can be estimated by the numbers of white (and, rarely, of red) blood-corpuscles admixed with the mucus. A plentiful admixture of white blood-corpuscles to the mucus gives this a purulent character which may frequently be recognized macroscopically.

The mucus often shows a hemorrhagic discoloration, even with only superficial inflammation of the intestinal mucous membrane. Greater admixture of blood to the intestinal discharges almost always indicates ulcerative processes in the intestine or rupture of continuity of the mucous membrane in the rectum (fissures, abrasions, hemorrhoids). Here it need

only be mentioned that profuse hemorrhages, even without ulcerative processes, may take place in the gastrointestinal canal; for example, in hepatic cirrhosis and in thrombosis and embolism of the mesenteric artery.

The causes of endemic and tropical diarrhea, of many acute and chronic catarrhs, and of inflammations of the intestinal mucous membrane are to be found in the protozoa in fresh intestinal mucus or that which has been kept warm. Some forms of *dysentery* belong to this category, also *ameba enteritis, coccidia* and *infusoria diarrhea.* In his article, "Dysentery and Ameba Enteritis," which I should here like to refer to,[1] G. Hoppe-Seyler has described these conditions. But I cannot here enter upon the bacteriologic finding of the specific agents of many infectious diarrheas and intestinal inflammations, neither can I enumerate the parasites of the intestine, the existence of which can be determined by an examination of the feces.

Treatment.—The treatment of diarrhea and of acute intestinal catarrh must be instituted with a search for the cause. If it has been found that abnormality of the intestinal contents has produced the affection *the first indication in the treatment is the removal of the deleterious intestinal contents.*

As a matter of fact the action of the organism is based on the same principles: Diarrhea, produced reflexly, is an attempt on the part of the over-irritated organ to relieve itself, and this is further promoted by the increased excretion of fluid and the inappetence, also reflexly produced, which prevents the placing of additional labor upon the digestive organs, but gives them rest; thus a reserve power of resistance is produced. In fact, many diarrheas and catarrhs are self-limited and naturally run a rapid and favorable course; for this reason we should not interfere with the organism in its attempt at self-aid: *Primo die quiescere satis est; neque impetum ventris prohibere* (Celsus).

We aid this endeavor of the body by rest in bed and the administration of non-irritating drinks which immediately cleanse the stomach and the upper part of the small intestine and, after absorption, also the tissues of the body, especially the blood and the kidneys, thus compensating for the loss of fluids and the diarrhea by maintaining diuresis. By washing out the colon from below a large quantity of its contents can be more rapidly evacuated than by the diarrhea, and thereby we limit the absorption of abnormal products of decomposition, and give the irritated organ longer periods of rest, even if we do not always by these means succeed in rapidly controlling the diarrhea.

Warm infusions of herbs which contain ethereal oils (phenol, anise, chamomile, or peppermint) long enjoyed a great reputation in diarrhea,

[1] See "Modern Clinical Medicine," "Infectious Diseases," D. Appleton & Co., 1905, p. 681.

for the reason that they taste better than warm water, and as carminatives apparently have a mild action; but they may well be substituted for by warm mineral water with a small addition of table salt, by weak warm or cold tea, and, in some cases, by dilute rice water, barley water, oatmeal water or albumin water. When these drinks are to be taken for several days the last named deserve preference, as they simultaneously contain a minimum of nourishment.

The quantity of fluid that is to be administered depends upon individual conditions; it should be sufficient to quench the thirst, but should not be given in excess.

For intestinal irrigations, besides weak infusions of peppermint, chamomile, anise, etc., the most suitable fluids are saline solutions one-half to three-quarters per cent., under some circumstances with the addition of 0.5 to 1.0 of salicylic acid to the liter of fluid. Other disinfectants may be used for this purpose; some of them are not very effective as the solution must be very weak in order not to injure the intestinal epithelium, and in choosing these remedies their absorption in the rectum and colon must be considered. The efficacy of the washing depends largely upon the temperature of the fluid, which should be of the same temperature as the body, and the *quantity* employed must depend upon individual circumstances. I advise that the patient be placed in the left lateral posture, under some circumstances the pelvis to be raised, and as much of the lukewarm fluid to be introduced with low pressure (one-half to one meter) as can be borne without discomfort. As soon as there is a desire to evacuate the bowel we must suspend the irrigation and the patient should be permitted to discharge the fluid injected. In children from 200 to 500 c.c., in adults from 1 to 1½ liters and more may thus be used.

According to the indications of the case, in diarrhea we may irrigate the intestines morning and evening, or even three times a day; for example, in acute diarrhea combined with great tenesmus. In other instances a single washing of the intestine in the morning or in the evening will suffice. In so-called *nocturnal diarrhea,* which invariably occurs at night and, as a rule, in the second half of the night or early in the morning, it is advisable to give a chamomile injection in the evening, shortly before supper, or half an hour after going to bed. Such evening injections are, at the same time, the most effective remedies for peristaltic unrest of the intestine which prevents or interferes with sleep.

The best instrument to employ is an irrigator or glass funnel with a conveniently long rubber tube; but syringes may also be employed if a piece of rubber tubing 20 to 30 cm. in length is inserted between the syringe and the rectal bulb to allow for a possible recoil or jerk of the syringe, and to prevent trauma to the rectum. Care is necessary in the choice of an anal tube; the best are those made of hard rubber or

glass which have an olive-shaped bulb and are well and smoothly rounded off, and the rubber rectal tubes to be attached should have fenestra that have been smoothly rounded off. *The high introduction of longer tubes into the intestine is only advisable when the sphincter will not contract, and when we desire to dislodge an obstruction with the rectal tube;* for example, in a retroflexion of the uterus or in the case of perimetritic exudates. Even in the so-called *high* intestinal injection, a high introduction of the rectal tube is unnecessary; the flow of the fluid into the intestine is promoted much more by the position of the patient than by the introduction of a long tube: The knee-elbow position, the dorsal decubitus with the shoulders low and the pelvis high, perhaps even the placing of the patient upon a modern operating table which permits the maximum high position of the pelvis.

Occasionally we thoroughly cleanse the rectum and the sigmoid flexure, sometimes even the left flexure of the colon or the entire colon, by irrigating the intestine with a tube and funnel, similarly as in gastric lavage, raising and lowering the funnel and occasionally, after emptying the funnel, adding more fluid. The intestine, however, is more susceptible than the stomach, and the observance of, at least, the same precautions as in washing the other organ is necessary.

If the injurious intestinal contents cannot be removed by washing from below, it is advisable to try mild laxatives. The number of remedies to choose from is great, therefore individual indications may be considered. Young children bear calomel in small doses (0.01–0.03 several times daily) very well. In adults several (2 or 3) large doses (0.1–0.2) given at short intervals are sometimes of great value in diarrhea, although even with these doses [1] we must reckon with the frequent intolerance of mercury. In doubtful cases a good dose (20 to 30 grams) of castor oil is given, or rhubarb, cascara sagrada, laxative tea or lemonade (citrate of magnesia), a teaspoonful of Glauber salt, Epsom salt, sodium subsulphate or Carlsbad salt dissolved in about a quarter of a liter of water.

The *second indication* in the treatment of acute diarrhea and intestinal catarrh is to produce non-irritating intestinal contents. As a rule, we should be in no haste to bring about such a change. Fluids that are easily absorbed, water, tea, thin meat broth, rice water, and various mineral waters with a small addition of brandy or red wine are sufficient until hunger reappears or weakness becomes noticeable. In the choice of foods the results of the examination of the feces must be considered, and at first those foods must be avoided which coproscopy shows to be

[1] I particularly desire to emphasize that the belief that calomel disinfects the intestine is erroneous. Schütz proved in his experimental animals that by calomel —in contrast with castor oil—the bactericidal properties of the intestine were inhibited, and Strasburger found more bacteria in diarrheic stools after calomel than in those produced by castor oil.

insufficiently digested or difficult of absorption. If we are dealing especially with fermentative diarrhea, those substances which produce fermentation—principally, therefore, the starches—must be at first avoided and an albumin diet given, or, mostly in fetid diarrhea, an albumin diet should be avoided or should be permitted to but a very limited extent, and carbohydrates given instead, perhaps in the form of gelatinous soups, milk soups, or cream soups.

In the acute diarrhea of little children, albumin water, thin gelatinous soups which are made with water, or veal broth may for a few days substitute for milk. Young infants should have a wet nurse, or, if this cannot be arranged for, sterilized milk suitably diluted with oatmeal gruel is the best food. For larger children racahout and other fine starches (arrowroot, avena, maizena, mondamin and rice flour) with milk, or milk and oatmeal gruel, may be given. A gelatinous mixture prepared by toasting equal parts of rolled barley and candied sugar and then boiling for a long time in water is very concentrated, possesses a high nutritive value, is easily taken, and well borne. The quantity of food is to be cautiously increased, as well as the concentration of the milk and other foods; for a time, however, we adhere strictly to the calory requirement of the youthful organism, and avoid overloading the intestine.

In adults also we must carefully regulate the nutrition; generally, however, they may more rapidly return to the ordinary diet than children.

Treatment by drugs is indicated when a strict diet combined with rest and intestinal lavage has proven unsuccessful. It is a great mistake to resort to drugs at the onset of the malady. This is, above all, true of *opium,* which is the best remedy with which to lessen or inhibit intestinal activity. If this is done while noxious material is still in the intestine, the organism is not only deprived of its natural power of self-aid by increased peristalsis, but it is damaged and the course of the disease is thereby protracted.

I believe it advisable to give such a dose of opium as is just sufficient to quiet the motor and secretory irritability of the intestine, and to repeat this dose at proper intervals. Occasionally larger doses may be given at night to secure rest for the patient.

A direct *anti-irritant* effect is produced upon the mucous membrane by gelatinous fluids: Gum arabic, decoction salep, decoction of althea, and also emulsions of oil and milk of almonds. These substances may be administered even with the addition of opium. In catarrh of the rectum and of the lower colon we may also employ these directly as injections; *enemata of starch* (retained enemata), perhaps with the addition of a few drops of opium, have a remarkably soothing effect upon the colon; they mechanically protect the membrane as does mucus, and do not decompose—at least, do not become fetid—and even appear directly to counteract decomposition.

Oil and the *preparations of bismuth* also mechanically protect the bowel if in proper suspension in water or oil, and injected as small, retained enemata. When there is painful tenesmus and spasm of the anus the local application of opium or other antispasmodics (extract of belladonna) in the form of suppositories is indicated; sometimes a simple suppository of cacao butter is sufficient, for the melted cacao butter covers the irritated mucous membrane with an oily surface, and protects and quiets it.

Anodyne and chemical effects are produced in the intestine by *alkalies* and *alkaline earths,* as they neutralize the fatty acids in the small intestine. The fatty acid salts irritate the intestinal membrane much less than free fatty acids. This explains the constipating effect of *chalk water* and calcium carbonate, as well as the anodyne property of Hufeland's children's powders (pulv. magnes. c. rheo.) which, in small doses, allay abdominal pain, expel gas, and regulate the bowels, but in larger doses have a laxative effect.

As *constipating remedies* antidiarrheics and astringents are still frequently resorted to, particularly tannic acid and tannic acid combinations, both by mouth and as enemata (tannin, tannalbin, tannigen); also the vegetables containing tannic acid (lign. campech., colombo, ratanhia, catechu, whortleberries, red wine, cocoa, acorn, acorn cocoa); the metals, above all, bismuth (bismuth subnitrate, bismuth salicylate, bismutose), lead acetate, and silver nitrate are valuable. In acute cases, however, we usually refrain from using the last named remedies.

It was supposed for a time that a disinfectant action upon the intestinal contents could be produced by creosote, salol, naphthalin, benzonaphthol and numerous other combinations from the group of creasols and naphthols. I have seen no noteworthy results from these or other intestinal antiseptics, or from calomel in large doses. Limitation of fermentation and decomposition in the intestine is best attained by the application of the principle of asepsis; namely, the intestinal contents which have begun to decompose are expelled from the intestine as thoroughly as possible without producing irritation therein, and until this cleansing process has been accomplished the ingestion of food—for a few days—is to be limited to easily absorbable material which will leave but little residue, and, if possible, to a fluid diet: Tea, meat broth, gelatinous soups. In a few days an increase of food may be permitted without any danger of a relapse.

In conclusion, I must issue a warning against the artificial arrest of diarrheas and intestinal catarrhs due to the excretion of toxic substances from the blood into the intestine. Paramount among these is the diarrhea in nephritis, the arrest of which with opium and similar remedies may bring about severe uremia.

CHRONIC INTESTINAL CATARRH

Chronic intestinal catarrh, like the acute form, may appear as a primary and substantive affection. Frequently, however, it is either secondary or of a symptomatic nature.

Etiology.—The causes of idiopathic chronic intestinal catarrh are, as a rule, identical with those which produce acute catarrh except for the difference that the baneful agents mentioned here act not only once and intensely, but that this effect is produced repeatedly or continuously. It happens by no means rarely that the chronic form develops from acute enteritis; that, therefore, also a single and transitory but active poison, an indiscretion in diet, severe refrigeration, or an infection, may be the cause of chronic intestinal catarrh. Above all, I desire to refer to the chronic catarrh of the colon which persists after tropical diarrhea and tropical enteritis. To mention an example from the present time, our soldiers who returned from China without having had severe forms of dysentery (although quite a large number had persistent irritation of the colon which in the cases observed by me proved to be chronic colitis) showed impairment of health for a long time although they did not become chronic invalids. More frequently chronic intestinal catarrh sets in, without an acute stage of onset, as the consequence of oft-repeated and continuous injury to the intestine, by improper food and an injudicious mode of life, by the prolonged use of powerful purgatives, and by constitutional anomalies.

"*Morborum fere omnium causa est stomachi infirmitas*" is particularly true of the different forms of chronic intestinal catarrh. Not only does weakness of the stomach impair the bowel, but also that factor which has damaged the stomach. In this connection the poor condition of the teeth, their insufficient use in mastication, the incomplete admixture of the food with saliva, and the bolting of food must be again referred to. We must also consider all the functional disturbances and organic diseases of the stomach which influence the chemical preparation of the gastric contents, above all, gastric dyspepsia, achylia, and apepsia, which run their course with deficiency in hydrochloric acid and pepsin. To these must be added chronic catarrh of the stomach, hypermotility which causes the chyle to be too rapidly propelled from the stomach into the intestine, and gastric atony, in which condition the chyle of the stomach undergoes an abnormally acid fermentation, and thus enters the bowel. Even gastric ulcer, which, by HCl-hyperacidity and hyperpepsia, makes gastric digestion so rapid, and also by HCl-hyperacidity reflexly stimulates pancreatic digestion, may induce chronic colitis as a complication if the chyle (which should be wholly absorbed and thus leave but little nutritive material in the intestine for the bacteria of fermentation and decomposition) accumulates in the colon, and if constipation, which

almost invariably accompanies gastric ulcer, then irritates the mucous membrane. Necessarily, however, none of the previously mentioned gastric disturbances need secondarily damage the intestine, for, to a certain extent, and frequently for a long time, intestinal digestion may compensate for derangement of gastric digestion.

The same is true of those disturbances originating in the liver and the pancreas which I have already mentioned.

Pathology.—Pathologico-anatomical changes in the structure of the intestinal wall, and to a less extent tuberculous, dysenteric, typhoid and carcinomatous ulcers, the cicatrices (arising from the former) which produce stenotic tumors, and peritoneal adhesions which limit peristalsis, abnormally fix the individual intestinal coils, or produce changes in their position, and thus hinder the propulsion of feces; these changes are almost invariably associated with intestinal catarrh, which, while sometimes limited to the vicinity of the primary lesion, occasionally may be distributed over extensive areas of the bowel. In fact, any condition may be combined with an impediment to the propulsion of feces, whether this be due to organic changes in the intestinal wall, to functional disturbance of intestinal peristalsis (habitual constipation), or whether it is caused mechanically by pressure or external torsion upon the bowel (uterine and ovarian tumors, hypertrophy of the prostate, peri- and parametritic, perityphlitic, pericholecystitic, perigastric or pericolitic adhesions, indurations and pseudo-membranes), and may give rise to chronic intestinal catarrh. We must mention also as causes of chronic intestinal catarrh the *circulatory disturbances* which produce chronic venous hyperemia of the intestinal mucous membrane. Among the more common of these conditions is chronic intestinal catarrh from abdominal plethora which is the consequence of a sedentary mode of life. Furthermore, intestinal catarrh is common as the consequence of stasis of the portal vein circulation, in hepatic cirrhosis and constriction of the portal vein, in valvular disease of the heart and chronic myocarditis, in chronic diseases of the lungs and pleuræ, and in chronic simple and tuberculous peritonitis.

In conclusion, parasites now and then produce chronic irritation and catarrh of the intestine.

The *pathological changes* produced by chronic intestinal catarrh are chiefly manifested in the mucous membrane, but the submucous tissue and the muscularis are often also implicated, much more frequently revealing inflammatory hyperplasia than a degenerative atrophy.

The mucous membrane is hyperemic and shows cellular infiltration; consequently it is swollen, and covered with mucus or muco-purulent masses. In some areas it is grayish red, livid, rusty brown, or it may show a slaty gray pigmentation. The submucosa is thickened and infiltrated; serous transudation is evident. The connective tissue is proliferated; the gland ducts are often elongated, and occasionally denote cystic

degeneration from constriction. The muscularis is sometimes hypertrophied and thickened. In differentiating these conditions, however, the contraction of the intestine must be borne in mind, for, if this is increased, it readily simulates hypertrophy, and an abnormal distention of the bowel may be easily mistaken for atrophy of the various layers of the intestinal wall. At the ileo-cecal valve a chronic inflammatory thickening of all these layers, especially of the musculature, may bring about stenosis of the lumen, which resembles pylorie stenosis due to chronic inflammation and may be the cause of invagination.

The question of intestinal atrophy, especially atrophy of the intestinal mucous membrane, has been much discussed of late since Gerlach, Heubner and others designated intestinal atrophy as a frequent post mortem phenomenon. Whether a vital pathologic atrophy of the mucous membrane can be absolutely denied must be decided by renewed investigations as soon as possible after death.

Fatty degeneration of the muscular fibers, more conspicuous in the longitudinal than in the circular fibers, has been noted now and then in chronic intestinal catarrh, but, as a rule, it is rare; it has been described by Kussmaul, Maier, and E. Wagner. There are but few isolated observations of sclerotic and fatty degeneration of the intestinal nerves, the ganglia and fibers of Meissner, and of Auerbach's plexuses.

The functional disturbances of the intestine produced by chronic intestinal catarrh are prominent in the clinical picture; associated with these are disturbances of nutrition and blood formation as well as other constitutional symptoms.

Symptoms.—While in acute intestinal catarrh diarrhea is characteristic as a motor irritative factor, in chronic intestinal catarrh this is rare, and constipation is the rule. In explanation of this it has been assumed that, under the influence of pathologic processes in the intestinal wall in chronic catarrh, the muscularis of the intestine suffers functionally by the production of *atony,* or from an organic damage by degenerative *atrophy.* But with chronic catarrhal inflammation frequently no change can be observed in the musculature of the intestine. Therefore neither atony nor atrophy of the intestinal muscularis will explain the constipation in chronic catarrh. Nothnagel has assumed that the cause is sluggishness of the intestines, a lessening of the *automatic* activity of the nervous apparatus of the intestinal wall, due to the pathologic processes of chronic catarrh, especially to chronic venous hyperemia. But this decrease in automatic activity does not simultaneously decrease the irritability of the nervous apparatus of the intestine to external influences. This is evident from the periodical and spontaneous occurrence of brief or more prolonged diarrhea, produced by errors in diet, drugs or fecal accumulation. Besides, in chronic intestinal catarrh due to local irritation, not infrequently large or small portions of the intestine may assume a state of spastic

contracture in which tonic spasm of the muscularis is indicated by severe pain (colica spastica). This spasmodic contracture may cause the retention of fecal masses in the greater portion of the colon (spastic constipation).

When constipation is absent in chronic catarrh of the intestine, abnormalities of the bowel movements may be observed, and this is true even when the bowels are apparently regular. These abnormalities are shown by the composition of the feces, which are of pappy or semi-fluid consistence with an admixture of mucus, by the evacuation of undigested particles of food, and by an abnormally fetid odor in consequence of decomposition of the pathologic secretions of the bowel.

In other cases of chronic intestinal catarrh constipation and diarrhea alternate in a certain regularity and periodicity. Without apparent cause, after one or several days of constipation, diarrhea occurs spontaneously, continues from one to several days, then ceases, and constipation reappears. Nothnagel has compared this alternation of constipation with diarrhea to the variations of the Cheyne-Stokes respiration, and assumes that, in the cases in question, constipation was originally present and persisted until by stasis of the feces in the colon a condition of irritation was produced which resulted in diarrhea. The irritation ceased after the complete evacuation of the intestine, and constipation reappeared.

With these exceptions, *intercurrent* diarrheas are usually due to some deleterious agent which, by irritating the intestinal wall, increases the intensity of the catarrh; often nervous influences are the cause. *Prolonged* diarrhea, on the other hand, indicates a complication of the intestinal catarrh with ulcerative processes.

Now and then I have observed in chronic intestinal catarrh a peculiar abnormality in the fecal evacuations, which resembles the condition in ischuria paradoxa. A movement of the bowels occurred daily, even regularly, the feces being soft, and the patients never complained of constipation.

Nevertheless, fecal masses were retained in the intestine and gradually increased so that they formed large fecal tumors. Years ago, when I was an assistant in the Pathological Institute, I held an autopsy upon a case of this kind and found in the colon a fecal mass weighing 4 kilograms. In the sigmoid flexure, where the largest fecal tumor was situated, there was an extensive pressure necrosis of the mucous membrane, also pressure ulcers one of which had perforated. Up to the time of death the patient in question had daily evacuated thin stools which had been regarded as sufficient. Among other cases of this kind, my most recent one is interesting. It occurred in a medical student who had severe colicky pains and a most offensive diarrhea, the thin fecal discharges being sometimes involuntary and soiling his bed and body linen most disagreeably; he was treated in the Clinic, and had also been treated elsewhere unsuccessfully,

with opium. When I examined this unfortunate man, I found a fecal tumor of about the size of a child's head bulging from the rectum, the sphincter of which was open. Manually I removed as much of this as was possible without anesthesia. Five days later this procedure—although diarrhea was still present—had to be repeated because on the removal of the fecal tumor another formed. After this the pains and the accompanying disturbances of the bladder ceased, and the bowels were regulated by injections of oil. Field has described such a condition under the name of cumulative constipation, and Courtade as latent constipation. From its analogy to ischuria paradoxa, I believe that this might well be designated as *constipatio paradoxa,* that is, a retention of feces notwithstanding existing diarrhea. A very intelligent patient who suffered from this paradoxical retention of feces quite aptly designated her disease as *"constipating diarrhea."* The condition reminds us of some cases of chronic intestinal catarrh and constipation in which liquid bowel discharges follow the ingestion of fruit, the use of some laxatives and mineral waters, and also occur in a mineral spring treatment, but do not relieve the patient by a satisfactory emptying of the bowel.

The *pain* in chronic intestinal catarrh is usually insignificant; nevertheless there are *irritable forms,* particularly of catarrh of the colon—colitis muco-membranacea—in which the patients suffer greatly from pain and persistent colic, similarly as in nervous mucous colic. In catarrh of the colon the pains radiate to the small of the back, to the hips and to the thighs, and very often to the entire back, and are lessened or entirely relieved by the evacuation of the accumulated fecal masses. The latter is true not only of some cases of sciatica but also of other neuralgias.

In other instances the patients are greatly annoyed by periodic or long-continued *disagreeable sensations* in the abdomen, and these sometimes engender a peculiar, melancholic state of depression. These sensations are chiefly localized in the hypochondria or the region of the flexures of the colon, but are also noted in other areas of the abdomen and the pelvis (the designation hypochondriasis, now employed only for certain depressing psychical conditions, was originally applied to some localized affections in which the colon was presumed to be especially implicated). These disagreeable sensations appear to be due to movements in the colon which are manifested in the way described; that is, they are increased after the ingestion of food, before and after a passage from the bowels, while frequently the patient is at ease so long as the intestine is at rest, i. e., while there is constipation. Besides the formation of other chemically irritant products of fermentation and decomposition, abnormal gas production is probably the cause of increased intestinal unrest, and rumbling, gurgling, or splashing sounds are heard not only by the patient but by those about him. The patients feel as though the fluid contents of the intestine were forced from one part of the abdomen to another,

or as if the intestines were filled with gas which could find no vent. Patients with disease of the intestine who have become hypochondriacs often torment themselves with strange fancies as to the cause of these sensations and sounds; these imaginations correspond with the remedies to which they resort to ameliorate the condition, especially the dietetic penance.

In undernourished persons with a thin, flaccid abdominal wall, peristaltic movements of the intestine are often visible and palpable. In these cases inflation of the abdomen from gas is rarely absent; often it is general, at other times limited to individual portions of the intestine. The passage of flatus usually brings relief, but the opposite condition is most unpleasant to the patient. The accumulation of gases in the bowel causes much discomfort, disturbance of sleep, a feeling of unrest and anxiety, a sense of constriction, arrhythmia and intermittent pulse, cardiac palpation which is often combined with precordial distress and difficulty in respiration. Not infrequently intestinal gas becomes incarcerated in dilated coils of intestine, and produces intense colicky pains (wind colic from overdistention of the intestinal wall).

In other cases of chronic intestinal catarrh the abdomen is retracted, the colon in certain areas or in its greatest extent is contracted, may then be felt as a band, and is painful to the touch. Upon palpation the sigmoid flexure may usually be felt as a cylindrical band which rolls under the palpating finger. Sometimes only one portion of the colon is contracted while other parts are flaccid or even meteorically distended.

The *nutrition* always suffers in chronic colitis, but especially when the small intestines are markedly implicated and less so when the disease is chiefly confined to the colon. Catarrh widely distributed throughout the small and large intestines causes extreme emaciation and debility. The best known forms of this are atrophia infantum, pedatrophia, or tabes mesenterica, the sequel of chronic intestinal catarrh in children in which, in contrast with the enormously distended belly, the patients are emaciated and withered to a skeleton, and with their sad, deeply sunken eyes, small, senile faces, and dry, discolored, withered skin they present a pitiable picture of suffering and human helplessness.

In adults this general disturbance of nutrition is rarely so marked as in children; but I have repeatedly known middle-aged persons to succumb to intestinal catarrh and chronic diarrhea which were the results of tropical enteritis. The dirty gray discoloration of the skin in some persons with chronic constipation which accompanies intestinal catarrh is typical. This "*constipation tint*" may be readily differentiated from that of anemia, cachexia, jaundice, and also from Addison's disease. To what alterations in the composition of the blood it is due cannot always be determined with the ordinary methods of blood examination. Dyscrasic intoxication may be presumed, but cannot always be certainly proven.

Anemic conditions are found in chronic intestinal catarrh, especially when the property of absorption in the small intestine is diminished. While, however, in this condition of hyponutrition we are dealing chiefly with *oligemia,* in colitis there is a destruction of red blood-corpuscles, probably due to toxic products of decomposition, particularly when the condition is accompanied by constipation. Inspissation of the blood with an apparent increase of the erythrocytes to 6,000,000 or more and a corresponding increase of hemoglobin I have observed at various times in prolonged diarrhea due to chronic gastrointestinal catarrh; tetany as a complication was noted in only one case. Severe progressive anemia which was formerly referred to atrophy of the intestinal mucous membrane is comparatively rare in chronic intestinal catarrh. In such cases Eisenlohr described degeneration of a dyserasic nature in the posterior and lateral columns of the spinal cord which was very similar to that I found in Addison's disease.

The Urine.—In chronic intestinal catarrh the condition of the urine depends mainly upon the relation of intestinal absorption to intestinal excretion. In severe diarrhea the daily amount of urine voided may fall to that of transitory anuria; constipation does not decidedly influence the amount. Albuminuria occurs in disturbances of intestinal digestion, and is not infrequent in abnormally increased decomposition in the colon; in fact this latter condition plays a rôle which is not to be underestimated in considering the etiology of nephritis as well as that of many diseases of the liver and the blood.

Disturbance of gastric digestion is rarely absent in chronic intestinal catarrh, often being the cause of the latter, and frequently its consequence. The gastric dyspepsia resulting from intestinal catarrh appears to cause greater subjective disturbance than a primary gastric affection. In many cases it is difficult to say whether the digestive symptoms, such as loss of appetite, a disagreeable taste, a coated tongue, pressure and fulness in the abdomen, discomfort after eating, eructations, flatulence and nausea, are to be attributed to the stomach or to the intestine. I believe a coated tongue is more frequently due to intestinal than to gastric disturbance, and most often results from constipation.

Various *nervous symptoms* are to be regarded partly as reflex phenomena from the intestine, and partly as due to the absorption of toxic substances formed by abnormal processes of decomposition in the intestine. To this category belong the oft recurring headache, the migraine and other forms of neuralgia, vertigo, psychical depression, disinclination for mental or physical exertion, a feeling of weakness and lassitude, drowsiness or insomnia, arrhythmia and intermittent pulse, tachycardia, as well as tetany and the other spasmodic conditions which are the consequence of constipation and which are especially observed in small children and feeble, easily irritated persons.

The appearance of fever in chronic intestinal catarrh indicates a complication with ulcerative processes and the implication of the peritoneum in the inflammation.

Diagnosis.—The diagnosis of chronic intestinal catarrh can be based only upon the results of the examination of the feces, for the history, the subjective symptoms, and the general phenomena, even the clinical investigation of the abdominal organs, are not conclusive; *the demonstration of mucus in the feces is alone decisive.*

The fundamental laws for the examination of the feces and their application have been described in the preceding sections of this article. They are generally applicable, and therefore may be employed in chronic catarrh of the intestine, at least in that form running its course with diarrhea. To complete my description, however, *I must enter somewhat more minutely into the condition of the feces in chronic intestinal catarrh which runs its course with constipation.*

In this affection the bowel movements are often found to be immoderately large. From this fact we may differentiate copropoesis or polycopria, provided these large masses are regularly voided for a long time; where this is not the case, but small portions and then occasionally very large ones are passed, the evacuations are insufficient and there is consequently fecal accumulation.

When the fecal masses voided are invariably large—large in comparison with the ingested food—there is poor assimilation, insufficient absorption in the small intestine, and profuse excretion of digestive juices and pathologic products from the intestinal wall. Accurate investigation of the feces for undigested remains of food permits the recognition of their nature and the seat of the disturbance, and simultaneously indicates, as has already been stated, the advisability of testing the function of the stomach, and also, perhaps, the glycogenetic function of the liver (alimentary glycosuria). A decision as to the *proper quantity* of the feces, aside from their form and composition, requires much practice (repeated weighing of the total solids of the feces, which is alone decisive, is impracticable) and presupposes an exact knowledge of the previously ingested food, its quantity, composition and preparation. We have here, it is true, a constantly varying but, nevertheless, in its composition and nutritive value, an average diet (already referred to in my "Text-Book of the Diseases of the Digestive Organs"), a similar form of nutrition for all patients under observation until, from the requirements of the individual case, deviations from, additions to, restrictions in, or substitutes for this normal diet become necessary. As the feces of all patients are examined separately from day to day, and are preserved for subsequent and necessary tests, it is easy to determine pathologic change in a bowel movement, any alteration in the form, the consistence, or quantity, also any abnormal admixture; even individual peculiarities may be at once recognized. I believe it to be important to

resort to coproscopy in every case, not only once or occasionally, but regularly and until in a given case we have arrived at a definite conclusion in regard to the digestive processes, not only the intestinal but also the gastric, not only the digestibility of the chief articles of food but also of a great number of foods. This method of clinical observation, of the study of the ingestion of food, and of the examination of the excreta, which, in the main, I learned from my revered master, Kussmaul, and which I have elaborated systematically, has been for years of such service that the use of Schmidt's test diet to test the function of the intestine has become unnecessary. My method has the additional advantage that it can be adhered to for a long time, i. e., as long as necessary, while the other method is practicable for only a few days. I do not, however, mean to disparage the importance and value of Schmidt's teachings, and advise the employment of his test diet whenever the study of the nutrition and the investigation of the digestive processes cannot be carried out in the manner I have indicated. I also advise the physician not to be content with a single examination, or a single analysis, of the feces in order to make a diagnosis. The demonstration of connective tissue, meat, fat, or mucus in the feces aids us materially in differentiation, just as does the demonstration of albumin or sugar in the urine, or of bacilli in the sputum. But, as a matter of fact, such demonstration points out only where and how subsequent clinical researches are to be begun and further developed.

By a fecal examination we are enabled to recognize, in the given case, whether the chronic catarrh is localized in the small intestine or in the colon. Most frequently the colon alone is attacked, or the colon simultaneously with the lowest portion of the small intestine.

From the appearance of the intestinal discharges, as well as from the subjective symptoms, various types of chronic (colon) intestinal catarrh may be differentiated: Flaccid, atonic or asthenic forms, and nervous, irritable forms. The first usually run their course with constipation; in this atonic constipation the bowel movement is dry and compact, and consists of adherent clumps or cylinders of immoderately large caliber, also of isolated, irregularly formed particles of varying size, and of fecal balls from the size of a nut to that of an apple. These fecal clumps are not always formed in the rectum (in the ampulla of which they may attain their greatest bulk) or the flexures, but often much higher, in the colon or even in the cecum. In chronic catarrh of the colon, running its course with atonic constipation, the abdomen is, as a rule, uniformly distended. On the other hand, when the entire colon is atonic and filled with gases and feces it is asymmetric, and only partially so when but a portion of the colon is diseased. If the abdominal walls are thin, the accumulated fecal masses may be felt in the flexures or the cecum, and also in other portions of the large intestine, as doughy tumors, in rare cases as hard, globular or cylindrical tumors, and in the colon they may frequently be

displaced toward the anus. These atonic forms of constipation and catarrh of the colon are found at all ages, not rarely even in children. Occasionally the impacted fecal masses may cause very painful attacks of colic.

The irritable forms of chronic catarrh of the colon are characterized by a tendency to diarrhea and colic. But many cases of irritable catarrh of the large intestine run their course with constipation of that type I have described as *spastic constipation.* In these cases, an irritation affecting the motor function of the muscularis of the colon, a spastic contracture which implicates large portions of this organ, causes the retention of the compact fecal masses, and constricts and compresses them; or the subsequent peristalsis in high portions forces the compact mass through the spastically contracted portions of the intestine. In this spastic constipation —which may also occur without colitis—the composition of the feces is extremely characteristic. The masses are cylindrical and of small caliber, often only of the thickness of a lead-pencil or of the little finger; sometimes they are very long, and in the chamber they assume a spiral shape like the shell of a snail. Sometimes, however, they are so short that their total amount appears to be wholly insufficient. Balls of small caliber, masses resembling the feces of sheep, are sometimes formed in the haustra but are not always characteristic of spastic constipation, for they may be discharged simultaneously with fecal cylinders of large caliber, or may be superficially embedded in these.

In some cases of colitis the types of constipation which have been described appear side by side, and atony and spasm in different areas of the colon may alternate.

Mucous Colic.—The severest form of catarrh of the colon, and at the same time the most irritable, I shall designate as *colitis muco-membranacea.* The views of physicians in regard to this common and stubborn disease have for a long time been far asunder, as is best proven by Whitehead's report: Of 120 authors who have written concerning it, less than 6 have given it the same name. Some lay the greatest stress upon the intestinal symptoms, others upon the nervous phenomena. Cruveilhier, Trousseau, Habershon, Leube and others emphasized its association with other inflammatory affections of the colon, especially with chronic catarrh; v. Leyden, Guttmann and others assume that the condition is due to a specific inflammatory process similar to that which is generated in croupous chronic bronchitis (Litten) and in dysmenorrhea membranacea (Wernich). In contrast with this, many other authors believe the affection to be a neurosis of the bowel, probably a secretory neurosis accompanying other general neuroses. Nothnagel, who was later supported by Ewald and many other adherents, especially emphasizes that the affection chiefly attacks nervous and hysterical women and hypochondriacal men, these presenting the clinical picture of colitis muco-membranacea. In accordance with this conception, Nothnagel proposed that we discard the names colitis and enteritis

for this affection, and substitute the designation *colica mucosa, mucous colic.*[1] In attempting to relieve or cure colitis membranacea, while correcting the general nutritive disturbances and regulating the intestinal functions, I have so frequently seen the accompanying nervous symptoms of this disease also improve or entirely disappear as to be convinced that these nervous symptoms are in many instances merely secondary and symptomatic, and in the overwhelming majority of cases the actual clinical picture of colitis membranacea is caused by a severe inflammatory affection of the colon, a malady which originates in the colon itself, or in its immediate vicinity and in some manner, by way of the peritoneum, attacks the colon. Nevertheless cases remain in which only a colica mucosa, therefore a sensory and secretory neurosis of the intestine, is present; however, these cases in my experience do not form the rule.

Colitis muco-membranacea may occur at any age; it is most frequent in middle life, rarer in childhood and in the aged. Women are most subject to the disease, particularly such as suffer from enteroptosis, dysmenorrhea or other sexual ailments; sometimes these develop after abdominal operation, perhaps in consequence of adhesions.

In the periods between the attacks most patients present the symptoms of simple catarrhal colitis with constipation. Periodically these symptoms are intensified by increasing constipation, and culminate in a typical attack of colic, in colitis muco-membranacea, at the acme of which a passage of mucus occurs; this must be regarded as critical inasmuch as subsequently, and particularly after a profuse evacuation of feces, there is rapid and conspicuous improvement, and even relative euphoria for some time.

The colic begins with a feeling of constriction, of burning or rawness in the epigastrium, the left hypochondrium, or the left lumbar region; this gradually increases to severe spasmodic or neuralgic pain of varying intensity which continues for hours or even days, being sometimes accompanied by reflex phenomena of diverse character (spastic constipation). In some cases vomiting occurs during this time; also, although rarely, slight rises in temperature and albuminuria; often we note palpitation of the heart, pulsation of the abdominal aorta, and a rapid, tense pulse. Finally, there is an evacuation of the bowels in which the quantity and composition of the admixed mucus is conspicuous. Often mucus alone is discharged.

The mucus discharged reveals nothing especially characteristic since in simple catarrh of the colon also mucus may be passed in the form of shreds, tubes, globular or grape-like masses. But in colitis muco-membranacea the composition of the mucus is significant; it is hard and tough, much coarser than that usually discharged in intestinal catarrh, is membranous, and not dissimilar to croupy membranes although it con-

[1] See the article by Hoppe-Seyler in this volume.

tains no fibrin. There is unquestionably no desquamation of the intestinal mucous membrane, as was formerly assumed by some authors. On the contrary, a process of coagulation causes the peculiar change in the mucus. Intestinal epithelia and round cells in great numbers adhere to these mucus shreds. Only when these are absent, and when in the intervals free from attack no signs of intestinal catarrh are apparent, when, therefore, no mucus is discharged, can a purely nervous enteropathy, in the sense of a secretory neurosis or an enteralgia, be considered.

We should never be content with the proof of a catarrh and the presence of accompanying nervous symptoms or sequels of a colitis, for, in most cases, especially the severe ones, the diagnosis is only confirmed when, by a minute study of the conditions and by a clinical examination, perhaps under anesthesia, the cause and the origin of the secondary colitis have been determined.

There are rare cases of colitis muco-membranacea in which the malady terminates with a single attack (Nothnagel); usually, however, the affection is chronic and very tenacious. Many authors believe that colitis muco-membranacea is extremely difficult to cure or even incurable (A. Clark). In my experience the prognosis is no worse than that of other chronic intestinal catarrhs, especially if treated in an institution where by persistent and suitable curative methods excellent results may be attained. It is true there are cases of severe secondary chronic colitis which can be relieved only by internal remedies, and are curable only by gynecological or surgical treatment.

Treatment.—The treatment of chronic intestinal catarrh is designed to regulate the *nutrition* and the *action of the bowels* so that any irritation which may produce or increase inflammation in the intestinal wall is prevented, and an opportunity is given the bowel gradually to recover its tone.

In regard to *diet* it is very difficult to establish general rules, as the peculiarities of each individual case must be borne in mind. The food products which are easily digested, have a high nutritive value, and leave but little residue, are very numerous. We must discriminate from among these, and decide which are best borne by the diseased intestine, which, at the same time, will sufficiently maintain the normal strength, or so improve a reduced or weakened organism that it may again become strong. In many cases, especially such as occur in the debilitated, which run their course with marked irritability of the intestines, it is advisable to begin the treatment with a few days of rest in bed and liquid diet. As soon as possible, however, and while avoiding anything that will demand too great exertion of the digestive organs, I give the previously mentioned normal diet—a mixed diet in which special preference is given to foods cooked with flour—since by the amount and composition of the feces these permit us most easily to recognize deviations from the normal. If, after

investigation by a functional test of the stomach and the examination of the feces, disturbances in digestion become apparent, these are combated by the administration of hydrochloric acid, pepsin, pancreatin or pankreon, and papain, and, if these are unsuccessful or insufficient, changes in the diet must be instituted to prevent the undigested and unabsorbed constituents of the food from being propelled into the intestine, where they may produce irritation or processes of fermentation and decomposition, or maintain these. Abnormal decomposition necessitates a limitation of albuminous diet, particularly of meat; abnormal fermentation, a limitation of carbohydrates and foods containing cellulose, and the corresponding substitution for these of other foods.

The irritative forms of chronic colitis—such as run their course with diarrhea or spastic constipation and colic—require a diet which lessens irritation. In the torpid, atonic forms of intestinal catarrh, foods which mechanically or chemically stimulate peristalsis are to be employed, provided that by their use we do not over-irritate the intestinal mucous membrane.

As the type of a diet which will lessen irritation, the *dietary employed in ulcer,* in gastric ulcer, may be mentioned. But it must be remembered that an exclusive milk diet is less well borne in intestinal disease than in gastric disease (some forms of tropical diarrhea, however, form an exception to this rule, and are occasionally cured by a milk diet). Therefore, when milk is not well borne we modify the diet, and give milk diluted with oatmeal gruel or barley water, cooked with fine flour or with one of the numerous infant foods, racahout, hygiama, oatmeal, cacao, etc., or with the many mixtures of flour found in commerce, these to be given in a fluid or pappy form, and then, step by step, we pass to a more abundant diet suited to the individual case; for a time, however, mechanical preparation of the food must be insisted upon (disintegration with a meat chopper).

In diarrhea some foods and fluids enjoy a certain reputation because they have a constipating effect; for example, barley water, rice, cacao, eggs, tea, red wine, whortleberries, and the juice of stewed pears. In the case of tea this is not always true. Neither is it with red wine and veal, and, in the main, not to speak of individual peculiarities, we may say that all foods appear to have a constipating effect which are dissolved and absorbed in the gastrointestinal canal, and therefore leave but little residue.

In contrast with this there are many foods and luxuries which stimulate the intestinal function, increase peristalsis, and even have a laxative effect, naturally varying with the individual. Cold water will stimulate, but much more so carbonated waters, lemonade, beer, grape juice, asti, and champagne. Sugar and drinks containing sugar (sugar water), honey, milk sugar, stewed fruits, marmalade, fruit jellies, etc., produce fermenta-

tion in the gastrointestinal canal and by the action of their acids stimulate peristalsis. The effect of buttermilk and of kefyr, of sauerkraut and many fruits, may be referred to their acid effect as well as to the development of gas. Besides the direct action of its acid, with sound fruit the indirect effect of its sugar must be considered. Sour wines and salad have a similar action, and the latter, besides, has the same property as many vegetables and contains numerous undigestible constituents (cellulose) which mechanically irritate the intestine inasmuch as they are not decomposed by bacteria, and thus they distend the intestine. The foods which *mechanically stimulate,* such as the heavy varieties of bread containing bran—bran bread, brown bread, ration bread (brown Tommy), Graham bread, Rademann's DK and Simon's bread, etc.—favorably as these act in atonic conditions of the intestine, are little indicated in the irritable forms of intestinal catarrh; for example, in colitis muco-membranacea and spastic constipation. The same may be said of unripe, unpeeled fruit, of the skins and seeds of grapes and many other fruits, of heavy varieties of cabbage and the legumes when insufficiently boiled.

Food rich in fat (cream, butter, fatty meat, fatty sauces and salads, farinaceous foods cooked in fat) in most persons stimulate intestinal peristalsis; generally, however, with the exception of milk, cream and butter, it is difficult to digest and is a standard for testing the so-called "digestive strength" of the stomach. Coffee, tea, tobacco, and brandy are judged according to their individual effects; the same is true of very salty or spiced delicatessen and the like. The irritation they produce in the intestinal wall varies; gradually, however, the digestive organs become accustomed to them, but subsequently the mucous membrane and musculature of the intestines, the liver, the vascular system, the kidneys and nervous system begin to suffer. Very few luxuries and delicatessen are suitable in dietetic treatment.

Artificial food preparations, I believe, should be used only occasionally, for, as permanent constituents of the dietary, food which is daily prepared afresh in the kitchen is preferable for the healthy as well as for the sick.

The same thought and discrimination should be exercised in regularly supplying the body with food as is devoted to the regular evacuation of the intestinal contents.

In chronic intestinal catarrh accompanied by diarrhea, regular and long-continued intestinal irrigations in the manner described are advisable. Besides lukewarm chamomile infusion or lukewarm saline solution, for this purpose mild mineral waters (Ems water, Carlsbad water diluted with an equal part of warm water) come into question, also dilute solutions of salicylic acid (1:1,000) or very dilute lysol solution (1 or 2:2,000). From solutions of tannin (5:1,000), of ichthyol, silver nitrate (0.5:1,000) and other solutions of silver I have, as a rule, seen no better results than

from suitable washings with chamomile water and mineral waters of proper temperature, provided the cause of the diarrhea was not in the lowest portion of the intestine. I have never employed long tubes for intestinal irrigations if the sphincter ani retained its tone. In laceration of the peritoneum alone are we justified in attempting to reach a point above the sphincter tertius, but in deep-seated stenoses we must try to pass the stenosed area by means of long, soft, rectal tubes.

These watery enemata may also be employed in chronic intestinal catarrh which runs its course with constipation and retention of feces. There is no disadvantage from their continued use provided the quantity of fluid is no larger than is necessary to produce evacuating peristalsis. But the frequent, or even regular, employment of so-called high intestinal injections of 2, 3 or more liters is not justifiable, and I have repeatedly heard patients state that their intestinal disorders, particularly of the atonic form, were aggravated by these forced injections. On the other hand, it cannot be denied that large injections into the intestine, if well borne, are particularly beneficial in cases of secondary colitis in which old peritoneal adhesions hinder the passage of feces. Inflammatory processes in the pelvis, even those which have run their course, in some women prevent the employment of even small injections on account of the pain they produce; that is, the quantity of water which can be injected into the intestine without causing pain is insufficient to stimulate an active peristalsis; the same is true in many cases of spastic constipation.

The most effective and, at the same time, the mildest treatment is by *oil enemata,* which Kussmaul and I employed in all affections of the colon running their course with constipation. Ten years ago I described their use, technic and physiologic effects (*Berliner klin. Wochenschr.,* 1893, Nr. 3 u. 4), and since that time the method has been extensively and most successfully employed in hospital and private practice.

The oil, lukewarm olive oil, poppy-seed oil, or sesame oil, if of proper temperature and carefully injected, penetrates high up into the intestine, similarly to the oil in the wick of a lamp. The passage of the oil is favored by placing the patient in a proper posture—usually the left lateral position or the dorsal decubitus with raised pelvis, under some circumstances even the knee-elbow position—by the pendular movement of the intestines, and by the physical composition of the oil (absorption).

Primarily the oil lessens the irritability in the intestine, perhaps acting like ointment upon a wound: It coats the intestinal mucous membrane and protects it from pressure and friction, loosens from it the mucus and adherent feces, limits the absorption of fecal fluid in the dissolved products of decomposition at the oiled areas of the intestinal wall, and thereby prevents a marked inspissation of the feces.

There is no special difficulty in retaining the oil in the intestine for hours. While so retained, free oleic acid is split off by the action of

bacteria, by the intestinal juices higher up in the intestine, and also by other undecomposed digestive juices, particularly by bile, and this causes a marked increase in the activity of the acid contained in the oil (0.34–3.97 per cent.). The oil which has beeome acid (simultaneously with the split-off glycerin, the soaps formed from the oleic acid, and the alkali of the intestinal juices) stimulates the bowel in a manner closely corresponding to intestinal peristalsis, and as soon as it reaches the necessary degree of acidity for a chemo-reflex eauses an evacuation. It also acts upon the excretion of bile and intestinal fluid, the oleic acid split off having a reflex action. Only a small portion of oleic acid is saponified and absorbed, for at least as mueh oil is evaeuated as was injected into the intestine.

The exceedingly mild effect of the oil may be explained by the theory that the oil in the intestine acts only as an evacuant after it has loosened the fecal masses from the intestinal wall and has rendered them pliable and smooth, so that a comparatively slight peristaltic action is sufficient for their expulsion. The undecomposed oil present in excess produces no further irritation of the mucous membrane, at least patients rarely complain of any sueh subsequent effects from the oil as follow the taking of laxatives; on the contrary, the evacuations, as a rule, promote their comfort. Diarrhea rarely occurs after enemata of oil, although, when the effect of the oil is at its height, evaeuations may oeeasionally take place several times during the day.

When the decomposed oil and the feces have been expelled from the colon by one or several evacuations, the previously described chemism again affcets the retained oil which adheres to the mucous membrane of the bowel. If the quantity of oil is small, a long time will be neeessary to stimulate peristalsis and to form oleie aeid, glycerin and quantities of soap. The number of evacuations will, however, become less until, finally, an additional amount of oil will be neeessary to regulate the bowels.

According to this view, and wherever indicated, a treatment with oil may be instituted whieh will meet the individual cireumstances of the case. This indication is found in *all forms of constipation,* particularly in such *as run their course with irritative symptoms,* but especially in inflammatory, eatarrhal and ulcerative processes of the colon immaterial whether these are aeeompanied by peritoneal—non-perforating—symptoms or not.[1]

The *dose of oil* employed in the enemata depends upon the size of the patient and the seat of the pathologic focus in the intestine. For adults the first injeetion should eontain from 200 to 400 grams, for ehildren 50 to 100 grams of oil at the temperature of the body; this is

[1] Other indications and individual points are given in the original article, quoted above, in the *Berliner klin. Wochenschr.*, 1893, Nr. 3 u. 4.

permitted to flow from an irrigator, or through a funnel or good syringe (not a bulb syringe) while the patient is in the left lateral posture—the bed or sofa to be previously protected with a rubber sheet or other suitable material—the syringe is then attached to the anal nozzle by means of a black rubber tube 20 cm. long, and the oil is permitted to flow slowly into the rectum. After the injection of the oil the patient remains in the same position for about a half hour, then for one or two hours longer upon his back, preferably in bed, until an evacuation occurs. Should this waiting be prolonged, or should the patient be uncomfortable from the prolonged retention of the oil in the intestine, which is apt to be the case when there is flatulence, the oil may be evacuated after four, six or eight hours by an injection of chamomile or anise water, or even with simple lukewarm water. These enemata of oil should be repeated daily until the intestine is empty, until all the old, hardened fecal masses are passed, or, still better, until the oil has had its maximum effect, which is signalized by the passage of thin, semi-solid feces, occasionally still containing undecomposed bile. Then a pause of one, two or more days is made until oil no longer appears in the evacuations, and the feces again show a tendency to dryness, or until an evacuation fails to take place daily.

There are cases in which a few enemata of oil, even a single injection, will permanently remove the difficulties due to fecal accumulation. But in chronic catarrh of the colon running its course with constipation the oil treatment, as a rule, must be continued for weeks or even months until the catarrh has been relieved as well as the functional disturbance. In these cases, after the first complete evacuation of the intestine an injection of oil is given regularly every day or on alternate days, and only after improvement appears is the oil to be used more rarely, perhaps twice or even once a week, until permanent recovery is brought about.

In such cases it is advisable to give the oil enema at night in bed just before the patient goes to sleep. We should begin with 200 to 300 grams, and subsequently use doses according to their effect: if the patient is disturbed at night by a desire to evacuate the bowels, less oil should be given the next time so that the evacuation may occur at a convenient morning hour. To secure this result small quantities of oil, 100 grams, 50 grams, or even less, are often sufficient. There are, however, cases in which the oil, while retained during the night in the intestine, fails to produce an evacuation in the morning, and we must then resort to a simple enema or other remedies. It cannot be denied that prolonged oil treatment sometimes produces disagreeable results, for example, the unconscious or involuntary passage of the oil. These disagreeable features, however, may be avoided by better technic in giving the enema, by proper dosage, and other slight precautions; where this is not the case we must return to the *use of laxatives* for the proper regulation of the bowels.

We differentiate between mildly acting (lenitive) and powerfully acting

(drastic) laxatives. In chronic colitis and other irritative conditions of the intestine mild remedies only should be employed, and these may be given in small doses for a long time without injurious effect. Chief among these remedies are rhubarb (powder, tablet, or pills) for adults and manna for children, also tamarinds, cortex rangulæ, senna, cascara sagrada, sulphur, magnesia, compound licorice powder and aloes in small doses. Among the drastic remedies: Scammonium, jalap, colocynth, and podophyllin are more rarely employed, and usually only as additions to increase the action of more mildly acting drugs. If, in stubborn cases, we are compelled to resort to more powerful laxatives, we add to the pill ingredients which lessen irritation, for instance, extract of hyoscyamus or extract of belladonna, both of which have an anodyne effect without influencing the peristalsis. In spastic constipation, atropin subcutaneously or, for long-continued use, in solution by mouth is very beneficial:

℞ Atropin sulphate..... 0.02
Aqua menth. pip..... 20.00
M. D. S.: Seven to ten drops three times daily.

Where no dyspeptic conditions complicate chronic intestinal catarrh and the stomach will tolerate it, excellent results are often obtained from the regular use of small doses of castor oil or olive oil given by the mouth. Whether the newer remedies, purgen, purgatin, exodin and others, are satisfactory for protracted use is questionable. Even California fig syrup has a highly irritating effect; at all events, it is not so mild as the name would lead us to suppose.

The aperient spring waters and the so-called neutral salts occupy a special position among laxatives: Magnesium sulphate, Glauber salt, sodium chlorid, magnesium carbonate and citrate, artificial and natural Carlsbad salt, and sodium hyposulphite. They not only promote fecal evacuations, but also the discharge from the intestine of non-absorbed constituents of the food, and cause an effusion of fluid into the gastrointestinal canal, an absorption of water, and the stimulation of the glandular elements of the mucous membrane to increased activity. Usually the artificial salt solutions are taken in too concentrated a form; therefore, on prolonged use, the epithelium of the stomach and intestine suffers from their hypertonic influence. For this reason, I employ solutions of salt only occasionally, and for long-continued use, and as an adjunct to the oil treatment, prefer the natural mineral waters.

The choice of a mineral spring depends upon the indications given by the functional test of the stomach and the intestine. The hot springs and heated natural waters (either alkaline, muriatic, or alkaline muriatic) by their heat lessen irritation, and therefore are rather constipating, while the cold waters, especially those which contain carbonic acid, have a

stimulating effect upon peristalsis. True spa treatment often fails to cure the severe forms of chronic colitis. I have, however, known great benefit to follow the treatment at Kissingen, Homburg, Tarasp, Carlsbad, Wiesbaden, and other resorts, provided the use of the waters was instituted after the most acute disturbances had been relieved by treatment in a sanatorium or at home, especially by the oil cure.

In the majority of cases of chronic intestinal catarrh of gastric origin, also in the numerous secondary complications which involve the stomach, systematic local treatment of this organ by lavage (early in the morning upon an empty stomach) is very advantageous. The effect of lavage upon the appetite, upon the functions of the stomach, upon the upper small intestine, upon the liver, upon the blood and the kidneys, and last, but not least, upon the morale of the patient is so well known that I need not enter into details; in diarrhea alone does gastric lavage occasionally have an unfavorable effect, and in these cases it should never be employed.

A number of other remedies indirectly influence the pathologic processes in the intestine and the disturbances in function dependent upon these. Heat, for example, is beneficial by lessening irritation (warmth of the bed and rest in bed); hot dry cloths, hot bottles, thermophores, moist heat by means of compresses (cataplasms) are all useful for relieving abdominal pain and colic. As a rule, patients with disease of the intestine and particularly those who suffer from diarrhea and whose nutrition has been damaged require a great amount of heat. They are easily chilled; when the physician advises them to wear an abdominal binder they follow the advice with excessive eagerness. In the winter there is no objection to wearing a flannel binder, but usually the wearing of these bandages does harm rather than good, since they weaken the skin which steadily becomes more susceptible to the effect of cold.

Cold applied to the skin reflexly stimulates peristalsis. Its hydrotherapeutic employment in cases of catarrh combined with intestinal atony produces remarkably good results; for this purpose Sitz baths of brief duration, bathing of the abdomen, friction, half-baths, douches, river and sea baths, may all be employed to relieve constipation. Besides, by improving the circulation, these procedures have a curative effect upon the intestinal catarrh which may even be increased by the addition of salt to the bath; therefore, salt baths, mineral baths and peat baths are advisable.

In suitable cases *electricity* may also be employed: The anode of the galvanic current for the amelioration of pain which originates from spastic conditions and neuralgia; faradization of the abdominal wall to stimulate intestinal movements.

Massage is indicated in some of the cases. When there are irritative conditions in the intestine and the peritoneum, pains, spastic constipation, and the presence of hard fecal masses in catarrhally inflamed portions of

the intestine, massage is contraindicated, while in the atonic and torpid conditions which mark some cases of chronic colitis brilliant results are sometimes attained by massage. Vibration massage is usually less harmful than manual, and is often well borne when the latter is contraindicated. We cannot, however, be certain of its advisability, for apparently, like many of these procedures, it presupposes more or less psychical susceptibility.

Medico-mechanical treatment and gymnastic exercises combined with spa and bath cures are very useful for the constipation of chronic colitis.

Besides these auxiliary methods of treatment, in the severe forms of secondary colitis (colitis muco-membranacea) which originate from inflammatory processes in the pelvis and abdominal organs, gynecological measures must be considered: Vaginal treatment, rectal treatment by bougies, under some circumstances even laparotomy for the purpose of breaking up peritoneal adhesions or the extirpation of tumors or diseased organs, for example, of an ovary, of the uterus, or of the tubes. I am not yet ready to relate my experiences with thiosinamin injections in cases of peritoneal adhesions which developed after abdominal operations, but the subject is a promising one.

III. INTESTINAL TUBERCULOSIS

Tuberculous infection of the intestines is caused by tubercle bacilli which reach the mouth in some manner and are swallowed. Frequently the infection is self-conveyed by the sputum of a tuberculous individual, who swallows it and thus introduces numerous tubercle bacilli into the intestine; but the tuberculous sputum of others may be directly thrown into the mouth of a healthy person by coughing, or it may be conveyed from the lips of a patient to a healthy person in the form of the finest particles. Dried tuberculous sputum in the air contains bacilli which upon inspiration do not all reach the deep respiratory passages and the lungs, but may adhere to the wall of the buccal cavity and be swallowed with the saliva. Little children playing with earth frequently introduce tubercle bacilli into the mouth with their dirty fingers as well as with toys and other articles which they lick or bite. From tuberculous wounds, with soiled fingers, instruments or any other object, children as well as adults may introduce the tuberculous poison into the mouth.

A mode of infection the importance of which was for a long time undervalued, and subsequently for a time exaggerated, is by means of food and drink containing bacilli (feeding tuberculosis). Since the discovery of the tubercle bacillus by Robert Koch, bacteriologic investigation has revealed a number of microörganisms resembling tubercle which at first were considered to be different in nature but are now known to be a more or less *common variety of one fundamental form*; they have developed by

adapting themselves to new conditions of existence. If, therefore, the tubercle bacilli of mammals, of birds, and of reptiles are considered to be identical, the importance of infection by means of food is greatly enhanced. But the view of v. Behring that "infant's milk," i. e., cow's milk given to nurslings as a substitute for mother's milk, "is the main cause of the development of tuberculosis" is far fetched; he should have said, the main cause of primary intestinal tuberculosis. Between the latter and pulmonary tuberculosis there is a wide difference. And although we cannot doubt that tubercle bacilli, like many other bacteria, may permeate the intestinal wall of the young without causing demonstrable changes at the point of entrance, yet such bacilli fail to reach the fluids of the body and to infect the lungs—in particular, as well as other organs—by means of the blood, but they infect the regional lymph-glands in those areas of the intestine which they traverse, and, after penetrating to the roots of the portal vein circulation, also the mesentery and the liver.

Primary enterogenous hepatic tuberculosis is very rare in man; primary enterogenous tuberculosis of the mesenteric lymph-glands—with or without tuberculous lesion of the intestine at the point of invasion—is, however, much more frequent, but, compared with pulmonary tuberculosis, is still relatively rare.[1] Enterogenous infection cannot, therefore, be the main source of (pulmonary) tuberculous development. Otherwise, we must assume that the bacilli not only pass unhindered through the intestine but also through the great regional lymph-gland apparatus of the intestine in the mesentery. That the latter is not the case is distinctly evident, but rather from the pathology of lymph-gland tuberculosis, especially tuberculosis of the mesenteric gland, than from the histology and physiology of the lymph-glands. I do not, however, deny the possibility that, as tuberculosis advances from the mesenteric lymph-glands, the pathologic process may implicate the intrathoracic lymph-glands and finally attack the lungs; but this occurrence is exceptional and by no means the rule. The same might be said of the primary lesion of tuberculosis in the lymphatic apparatus of the nasopharyngeal space, of tuberculosis of the pharyngeal and palatine tonsils and the lymphatics of the neck; but this digression leads us too far afield.

In contrast with the enterogenous development of intestinal tuberculosis, hematogenous intestinal tuberculosis in the form of miliary tuberculosis and tuberculous metastases is of much less importance. On the direct transference of the tuberculous pathologic process from a diseased organ to the neighboring intestine and its peritoneal coat, the primary focus of disease, as a rule (for example, a fungus coxitis, genital tuberculosis, or caries of the vertebra), becomes prominent in the clinical picture.

[1] The opinions of authors as to the frequency of primary intestinal tuberculosis are far asunder; the prevalence of tuberculosis among cattle varies within wide limits (7 to 45 per cent.) according to the region where this prevails.

PATHOLOGY

Pathologico-anatomically tuberculosis belongs to the most common of intestinal affections. We differentiate *primary* or *substantive intestinal tuberculosis* from the *secondary* form which occurs in consequence of earlier tuberculous disease of other organs, particularly of the lungs. In point of frequency, secondary intestinal tuberculosis preponderates greatly over the primary form. A definite opinion as to the *absolute* frequency of primary intestinal tuberculosis cannot be obtained at this time even from the material collected by Heller, v. Hansemann, Lubarsch and others. Experience indicates, however, that *primary feeding tuberculosis*, which, besides primary intestinal tuberculosis, includes also primary tuberculosis of the tonsils and the lymphatics of the neck, primary mesenteric gland tuberculosis and primary (isolated) peritoneal tuberculosis, *is not a rare affection in infants and children.*

When tubercle bacilli collect in a tissue, an inflammatory focus develops and the chemical poisons generated by the bacilli at first damage and destroy [1] the preexisting tissue cells, by chemotaxis they cause an accumulation of leukocytes, and in the healthy tissue cells in the neighborhood of the port of invasion which do not as yet contain bacilli they produce a reactive inflammation. Soon, however, the descendants of the proliferative tissue cells, the epithelioid and giant cells, succumb to the action of the adherent poison produced by the bacilli, and simultaneously obliterate preexisting vessels and prevent the formation of new ones.

Thus the process of destruction advances farther and farther from the point of invasion.

Tubercle bears the stamp of an inflammatory granulation proliferation, and differs only from other granulations by having a nodular form and by remaining without vessels (Ziegler). In the main, the morphologic changes in the tissues produced by tubercle bacilli are of varying kinds; simple exudative inflammation, suppuration, and the formation of granulation tissue and exsudation may combine in such different ways as to cause various anatomical pictures (Orth). Nodules may even be absent in the tuberculous inflammatory foci, in which case the decision as to tuberculosis can be made only by the tubercle bacilli.

The chief seat of the disease is the lymphadenoid apparatus of the intestine, especially the lower small intestine, the upper colon, and the region of the ileo-cecal valve. But any portion of the digestive tract, from beginning to end, may be affected by tuberculosis.

In the manner already described, *ulcers* may sooner or later develop from the tubercles of the solitary or agminate glands; these are either round, longitudinal, or irregular and sinuous. The ulcers spread prefer-

[1] *Karl Weigert's* "Shiwa."

ably in the direction of the vascular division, therefore vertically to the longitudinal axis of the intestine; thus the annular or girdle-like ulcers develop.

The borders of the tuberculous ulcers are usually infiltrated, the margins are elevated, and often undermined and permeated by the characteristic and more or less caseous and decomposed nodules. The latter are found also upon the base of the ulcer, which may be of varying depth, the floor being formed by the submucosa, the muscularis, and even by the serosa. At the autopsy we invariably recognize the seat of tuberculous intestinal ulcers even from without by the redness and thickening of the serosa and its permeation by small nodules. This circumscribed tuberculous peritonitis commonly favors the formation of adhesions and the extensive agglutination of diseased portions of the intestine with other organs covered by the peritoneum and the abdominal wall, and renders the perforation of tuberculous intestinal ulcers a relatively rare occurrence; when it does occur it is less dangerous because the succeeding peritonitis is sacculated. Where there is no serous covering of the intestine, as in the rectum, ulcerative tuberculosis leads to perirectal abscesses and subsequently to internal, external, and complete anal fistula.

Tuberculous granulation proliferation may terminate in caseation and ulcerous decomposition as well as in induration, according to the action of the bacterial toxins and the composition of the base on which they develop, and also perhaps the constitution of the patient (Ziegler). Hence we find in one case extensive and advancing ulceration in the intestine, in another, on the contrary, a conspicuous tendency to cicatrization and induration by connective tissue proliferation. Undoubtedly the cicatrization of a tuberculous ulcer must be regarded as a favorable process and indicative of a cure. This healing tendency may, however, in one sense be a disadvantage to the organism, for, as pyloric stenosis may develop in consequence of the cicatrization of a gastric ulcer at the pylorus, so by the cicatrization of an annular tuberculous ulcer of the intestine *constriction of the intestine* and severe disturbances in function may be brought about. Such cicatricial stenoses in the intestine occur simultaneously with tuberculous ulcers; I have repeatedly seen them multiple in the same case.

The most extreme constrictions of the intestine are the ring-shaped, cicatricial stenoses which originate from annular ulcers, and are very often short. The cicatrices which project spur-like into the lumen of the intestine and encompass only a portion of its circumference, at least in the small intestine, do not decidedly affect the permeability of the latter. Nikoljski draws the contrast between tuberculous and fibrinous tuberculous stricture which does not originate from an ulcer—consequently does not permit the recognition of cicatricial formation upon the mucous membrane—but arises from simple connective tissue proliferation in consequence

of tuberculous infection of the intestine—peribasillary sclerosis. A third form of tuberculous intestinal stenosis is the so-called hypertrophic stricture which is characterized by great thickening of the intestinal wall, a thickening which involves all of the layers, the mucous membrane by polypus proliferation, the submucosa and muscularis by hypertrophy, and the serosa by a decided connective tissue development which, by its permeation with masses of fat, increases the resemblance to a tumor. The differential diagnosis between carcinoma and tuberculosis may be perplexing even after extirpation of the stenosing tumor, until the microscope decides the question—as in one of my cases. In this hypertrophic form of tuberculous intestinal stenosis the submucosa with its vessels is most markedly implicated; some authors consider hypertrophic intestinal tuberculosis as of hematogenous origin (Nikoljski).

SYMPTOMS

The *clinical phenomena of intestinal tuberculosis* are manifold, and in the advanced stages very peculiar.

The initial stage of the affection almost always eludes observation, although it cannot be assumed that the inflammatory processes succeeding tuberculous infection of the intestine run their course without creating functional disturbances (pre-tuberculous diarrhea?).

From *indistinct, dyspeptic, intestinal disturbances* which in children, and not rarely in adults, are accompanied by a febrile movement, in the course of time, often very slowly, well developed clinical pictures are formed which correspond to the advancing pathologic changes in the intestinal wall and the mesenteric lymph-glands; these, in many cases, bear a relation to the symptom-complex of chronic intestinal catarrh with those anomalies in regard to the bowel movements which have been described. As a rule, however, just as in the pathological processes in the intestinal wall, ulcerative symptoms predominate. In every case peritoneal irritative phenomena may be superadded, either transitorily or permanently.

The symptoms of *subacute* and *chronic intestinal catarrh*, especially the increased excretion of mucus, may be due to the inflammatory process in the intestinal wall. Often, however, in compact as in diarrheic intestinal discharges, mucus is absent, while the intensity of other irritative symptoms does not permit us to assume the cessation of the inflammatory processes. Periodically mucus in greater masses is often admixed with the feces. In such cases the increased excretion of mucus is not always the sign of an exacerbating tuberculous inflammation, for functional disturbances accidentally produced by a long-existing ulcer, above all spastic contracture and fecal retention, may often produce in the mucous membrane of the affected intestinal area a great excretion of mucus, an irritative condition resembling colica mucosa.

In their nature and intensity the *ulcerative symptoms* of intestinal tuberculosis depend much less on the extent of surface and the depth of the tuberculous ulcerative process than on the intensity of the irritation of the exposed intestinal nerves upon the floor of the ulcer. If these are absent in ulcerated areas, it may happen that numerous and comparatively large tuberculous ulcers in the small intestine as well as in the colon run their course entirely latent, often causing no functional disturbance, while in other cases an isolated ulcer in the colon, if this implicate an important nerve branch, may produce the most severe irritative symptoms. The latter may be of a motor, sensory or secretory nature; to these must be added, as other symptoms of ulcer, hemorrhages and peritoneal and perirectal inflammations.

The motor irritative phenomena are most frequent in intestinal tuberculosis. *Diarrhea* is, therefore, the most common symptom of intestinal tuberculosis, and, when persistent, the possibility of intestinal tuberculosis must always be considered unless other symptoms indicate a different affection. In cases of tuberculous intestinal ulcers we now and then see typical spastic constipation combined with the previously mentioned painful evacuations of mucus which resemble mucous colic. The distress is often localized in the ileo-cecal region, and at first is usually attributed to an acute affection of the appendix. Violent tenesmus, often combined with the passage of blood, is produced by tuberculous ulcers of the rectum. In some cases the irritation of the rectum, and even that of the sigmoid flexure, simulates the picture of nervous hyperesthesia of the intestine. Small masses of dejecta which reach the rectum produce an irresistible desire to relieve the bowels, and this is frequently repeated at short intervals to the great distress of the patient. Since in these cases the evacuations are very small in amount, not rarely there is a paradoxical retention of feces, a constipation-diarrhea.

Peristaltic unrest accompanied by loud intestinal rumblings is an almost invariable symptom in the chronic diarrhea of intestinal tuberculosis; if, however, it continues after the diarrhea ceases, or is more marked during the periods of constipation, this increased peristaltic unrest is often the first sign of a beginning stenosis of the intestine. Added to it in the course of weeks and months are peristaltic pains which occur at more or less regular intervals, often every few minutes, with a characteristic tension of that portion of the intestine anterior to the obstruction. I shall later discuss this symptom of intestinal constriction.

The diarrhea of intestinal tuberculosis is frequently associated with pain; and the previously mentioned spasmodic state of the intestine, the contracture as well as the over-distention sometimes intercurrent with the former but also occurring without it, is often marked by excessive pain. During or before defecation, when hard feces pass through the ulcerated intestinal area, there is often true *ulcer pain* of great severity in the region

of the colon, especially in the lower portion. In such cases every evacuation distresses the patient for more or less time, often only for a few minutes, sometimes for hours, and then completely exhausts the sufferer.

Pain confined to definite areas of the abdomen, persisting for days but varying in intensity, and not markedly influenced by the intestinal function, may be regarded as of *peritoneal origin*, especially when there is local tenderness on pressure and a rise in temperature, and when nausea and a tendency to vomit are accompaniments.

These peritoneal symptoms may be acute and of great severity, and may be the forerunners of the gravest form of peritonitis, that due to perforation. But in intestinal tuberculosis an insidious onset and a course showing complications and frequent relapses is more common. Often the pain and the local sensitiveness to pressure are the only symptoms of the condition; sometimes a circumscribed, plastic, fibrinous exudate is formed, the demonstration of which may be impossible; after these peritoneal attacks adhesions of the intestine invariably form, and not infrequently cause new functional disturbances of the intestine. These are slightest in the very common serous exudates, often of enormous extent, which occur in tuberculous inflammations of the peritoneum.

Hemorrhage from the intestine is the most positive sign of ulcer. Tuberculous intestinal ulcers, however, rarely show a tendency to bleed, for the tubercle, as such, is non-vascular, and, furthermore, it is a peculiarity of the tubercular toxin speedily to obliterate small vessels. Nevertheless, in intestinal tuberculosis of considerable intensity and very difficult to control I have several times seen hemorrhage, particularly from the lower portions of the bowel. In such copious hemorrhages mechanical injury to the vessels from impacted fecal masses probably plays a greater rôle than erosion of the wall of the vessel—similar to the condition in pulmonary cavities—by a rapidly advancing process of ulceration. Yet in no other pathologic process is there such great recuperative power, such a disproportion between destruction and restitution, as is peculiar to the tubercular; consequently in severe and repeated hemorrhages from tuberculous intestinal ulcers we must never give up hope. I have seen the hemorrhage arrested, and even amelioration for years of all the severe anemic conditions which followed such hemorrhage.

The transformation of tuberculous granulation tissue into cicatricial connective tissue, connective tissue proliferation in the surroundings of tuberculous ulcers, and the formation from the plastic exudate of pseudomembranes and indurations upon the serosa of the intestine, corresponding to the seat and the areas surrounding the tuberculous ulcers, in some cases assist the conspicuous tendency of the latter to heal. These pathologic processes which favor the healing of ulcers may, however, have a decidedly injurious effect upon the permeability of the intestine. Cicatrization of an ulcer leads to cicatricial stenosis; diffuse infiltration and

thickening of the walls of a portion of the intestine prevent the propulsion of the peristaltic wave. Induration of the serosa of a section of the bowel also interrupts peristalsis, and pseudo-membranous adhesions readily produce traction and kinking in the lumen of the intestine, and consequent constriction and deformity which render the propulsion of the intestinal contents difficult.

By compensatory hypertrophy of the intestinal musculature above the point of obstruction these conditions may for a long time remain dormant and disturbances of fecal movement be compensated for. The diffuse, rigid infiltration and indurative thickening of definite sections of the bowel first become noticeable because these assume the form of a cylindrical strand or of a tumor of irregular shape and extent. These may then be palpated, but only in areas of the abdomen where the intestine and particularly the colon are readily palpable under normal circumstances, i. e., in the region of the cecum and of the sigmoid flexure. Often the beginning of the colon, the cecum with the appendix, and the ascending colon, are found to be the seat of the tuberculous infection. A tumor-like mass is felt in the right iliac fossa, and the sigmoid flexure in the left iliac fossa is found to be thickened, whereas, in many cases, these pathologic changes in the flexure of the colon, particularly upon the left side, cannot be determined during life.

In the small intestine, on account of the fluid or thin, semi-solid consistence of the intestinal contents, disturbances in compensation appear only late, after the intestinal constriction has reached an extreme degree. The same is true of the colon, particularly in the cases which run their course with chronic diarrhea. As, however, in most cases only a portion of the ulcer is cicatrized, while another is still ulcerated and irritated by the intestinal contents, every spasm due to the irritation of the ulcer, any accidental inspissation of the feces, may disturb compensation in stenosis of the colon.[1]

The first sign of a disturbance in compensation is the distention of the intestine above the stenosis. It may be circumscribed and soon disappear, or may rapidly increase in extent and distribute itself over the entire abdomen. Great peristaltic unrest combined with gurgling and rumbling in the abdomen then follows. With the increase of the abdominal distention the diaphragm is pressed upward, rendering respiration difficult and causing great discomfort. The distress is increased when nausea, eructations, singultus and a tendency to vomit appear, or when the accumulations of decomposed chyme from the small intestine regurgi-

[1] In a tuberculous stenosis of the ileo-cecal valve I saw a chronic invagination of the diseased lower ileum into the cecum and ascending colon. The replacement was comparatively easy, and subsequent invagination was prevented by a suitable bandage. Death finally occurred, not from intestinal occlusion, but chiefly in consequence of a complicating valvular lesion.

tate into the stomach and the eructations of gas or the vomitus acquire a disagreeable odor.

From time to time marked peristaltic waves occur in the greatly distended abdomen; and these may be due to extreme mechanical distention of the intestine, to chemical irritation from the highly decomposed intestinal contents, to reflexes, to the overloading of the stomach in eating or drinking, to cooling of the abdominal surface when the clothes are removed, or to mechanical irritation by percussion or palpation during the examination of the abdomen, particularly, however, from friction of the belly; these waves forcibly propel the fluid intestinal contents downward toward the obstruction, and so tensely fill a greater or less number of intestinal coils in front of the stenosis that their contours may be distinctly seen through the thin abdominal wall. These inflated intestinal coils are stiff upon palpation—*intestinal rigidity*—those nearest the stenosis often being as hard as wood, tumor-like. With this violent peristalsis agonizing pain, resembling uterine pain, sets in and gradually increases to the highest intensity. This intestinal rigidity and pain remain at their acme for a few seconds, occasionally for a minute, but rarely longer, and then suddenly cease, while the fluid contents, accompanied by loud rumbling and gurgling and apparently under the contraction of the overdistended intestinal coils (therefore antiperistaltically), are again forced back. Before this regurgitation occurs we occasionally hear in the stenosed intestinal area a murmur of long or short duration, a sort of squirting sound, which is due to the forced passage of the fluid and gas-containing intestinal contents through the stenosis.

During a peristaltic wave of this kind the pain is often so great that the face of the patient becomes contorted as in severe colic; the eyes sink in their orbits, the nose becomes pointed, and the expression of anxiety and suffering thus presented resembles the facies hippocratica. Cold sweat often appears, the pulse becomes small and weak, even intermittent; in very debilitated persons syncope follows.

Gradually the patient becomes so accustomed to this wave-like peristalsis that it no longer causes pain. This is not a good omen, for in such cases it certainly indicates that the tonus of the intestine has been impaired, that a great dilatation of that portion of the bowel situated in front of the obstruction has taken place, or there is regurgitation into the small intestine. Improvement in such cases is only apparent and transitory, and symptoms of intestinal occlusion rapidly appear.

In both varieties of intestinal tuberculosis, primary and secondary, the *local* symptoms which have been described are the same, that is, they *may* be the same. The *general clinical picture*, however, varies extraordinarily, and in secondary intestinal tuberculosis is influenced to a great extent by the tubercular infection of other organs, particularly of the lungs, to which intestinal tuberculosis is superadded.

Clinically the relations are frequently such that the first bowel symptoms occur only in an advanced stage, in pulmonary tuberculosis usually in the stage of cavity formation, and then profuse diarrhea, which is often of colliquative type, rapidly exhausts the remaining strength of the patient, who has been reduced by long-existing cough and expectoration, by undernutrition, by hectic fever and sweats. In such cases the intestinal symptoms are but slightly developed in proportion to the pulmonary findings. The ulcerative process in the intestinal wall often shows no correspondence with the severity and persistency of the diarrhea, for this is in part of toxic nature.

In other instances, simultaneously or very shortly afterward, the symptoms of tuberculosis of the lungs and of the intestines develop conjointly, and as *phthisis pulmonum et enterum* progress until the patient perishes. Even here the stubborn diarrhea is the most prominent of the intestinal symptoms. In a third series of cases, neither pulmonary nor intestinal symptoms are of special severity; the cough may be slight, expectoration entirely absent, functional disturbance of the intestine appears, but rather as constipation than as diarrhea; nevertheless the patients suffer from unconquerable loss of appetite, have the phthisical habit, and succumb to the slow but progressively fatal disease.

Among the clinical symptoms of another group of cases signs on the part of the tuberculous abdominal organs are prominent: Tuberculosis of the intestine and of the mesenteric lymph-glands, tuberculosis of the peritoneum, tuberculosis of the retroperitoneal lymph-glands and of the urogenital apparatus. In some of these patients the history points to a pulmonary catarrh and influenza, to a pleuritis, or to scrofulous disease of the tonsils or the glands, to bone and joint disease which may have existed previously; briefly to maladies during the course of which tuberculous poison has been conveyed to the abdominal and pelvic organs either by means of the lymph tracts or by the circulation. Where there is no such history we may be dealing with primary disease, especially with primary, intestinal, mesenteric and peritoneal tuberculosis in children and in the young. In these cases, as a rule, obstinately chronic remissions and improvements which simulate recovery are observed for years and decades, after which those changes in the intestine develop which lead to constriction of the intestine and to the formation of inflammatory tumors in the bowel.

These severe changes in the intestine and the peritoneum—without internal and surgical treatment—may spontaneously ameliorate. True, a new metastatic affection may appear in consequence of a severe infection from the intestine, and this is not rare, abscess may occur in the mesenteric lymph-glands, peritonitis may develop from perforation, or acute miliary tuberculosis may rapidly bring about a fatal termination.

Miliary tuberculosis, under the guise of a severe acute infectious disease, may set in, in most cases suddenly and unexpectedly, when from a

latent or manifest pathologic focus the tuberculous poison reaches the circulation and is disseminated throughout the body. Miliary tuberculosis of the intestine as such is usually masked behind an affection of other organs, for example, of the lungs, of the brain, and of the meninges. If, however, in consequence of a tubercular eruption of the mucous membrane and serosa of the intestine, the symptoms of diarrhea and of meteorism become especially marked, they may often resemble enteric fever.

DIAGNOSIS

The diagnosis of intestinal tuberculosis is easy when the symptoms of an affection of the bowel are added to demonstrable tuberculosis in other organs; when, however, they appear alone and no other signs of tuberculosis are present, they cannot always be recognized as of tuberculous origin, for chronic diarrhea and enteritis, ulcer of the intestine and constriction of the intestine, inflammatory ulceration of the bowel, and adhesive, exudative, perforative peritonitis develop also from many other causes. When, therefore, the chronic course of an intestinal affection, fever, sweating, the habitus, constitutional peculiarities, and, last but not least, the personal or family history lead us to suspect tuberculosis, we must seek to find the tubercle bacillus as the cause of the disease.

Sometimes the tubercle bacilli can be readily found by the ordinary methods, especially in the shreds of mucus and tissue which are voided with the feces. Very often, however, the most careful search for tubercle bacilli in the intestinal evacuations reveals nothing, and to this must be added the fact that when only isolated bacilli which are not adherent to the intestinal mucus are found in the feces these are by no means positive proof that the bacilli originate from a tuberculous affection of the bowel, for tubercle bacilli which have been accidentally swallowed may be present in the feces, and may be excreted unaltered.

If in such cases we cannot wait until the probable diagnosis is confirmed by subsequent observation, there is no danger in making an early diagnosis by the *cautious injection of tuberculin.*

Besides the clinical examination of the patient, the systematic investigation of the dejecta, especially its quality and quantity, and frequently also the examination of the rectum by palpation and rectoscopy, is absolutely necessary for the special diagnosis of the intestinal affection. The feces should be examined according to the rules given in preceding chapters. In addition, if we suspect the existence of tuberculous ulcers this suspicion will be confirmed by finding an admixture of blood and pus with the feces and a bloody discoloration of the mucus (Teichmann's or Weber's tests, spectroscopy). Furthermore, the exceedingly offensive odor of the flatus and the feces in the chronic diarrhea of phthisical patients is noteworthy and not without diagnostic importance; it adheres most

tenaciously to the bed-clothes and the body linen of the patient, and by the experienced physician is easily recognizable upon entering the sick room. Besides the functional weakness of the digestive juices and the limitation of intestinal absorption in consequence of increased peristalsis, a cause of this immoderate intestinal decomposition is the profuse admixture of pathological secretions from the intestine with the feces. Fat absorption suffers early and most severely in the chronic diarrhea of patients with intestinal tuberculosis, and to an extraordinary degree in those cases of primary or even of secondary intestinal tuberculosis in which the mesenteric lymph-glands are to a great extent diseased. Nevertheless, there is less fat in diarrheic feces than is found in steatorrhea with disease of the liver and of the pancreas.

Corresponding to this excessive intestinal decomposition we frequently find in chronic diarrhea from intestinal tuberculosis a decided increase of indican and of ethyl sulphuric acid ($C_2H_5HSO_4$) in the urine.

TREATMENT

The treatment of intestinal tuberculosis is exceedingly difficult, especially when it appears as a complication of tuberculous disease of other organs, and the obstinate diarrhea which accompanies it makes impossible the employment of one of the most important remedies in the treatment of tuberculosis, namely, hypernutrition. Recognizing this condition in pulmonary tuberculosis, we must be exceedingly careful to see that the sputum rich in bacilli does not find its way into the intestine by deglutition. Unfortunately, we cannot prevent small children from swallowing sputum; even with older ones it is difficult to enforce this prophylactic measure, as well as with very sensitive patients who regard the expectoration of sputum as a violation of estheticism which is more flagrant than their violation of the laws of hygiene by swallowing it. The educational effect of sanatoria for pulmonary diseases is especially evident in their recognition of the preventability of autoinfection.

In the *prophylaxis* of primary intestinal tuberculosis, certain sanitary laws in regard to the inspection of meat and the consumption of meat from animals infected by tuberculosis are operative. But a greater danger than confronts adults by the use of meat threatens nurslings and young children by the ingestion of milk from tuberculous cows. Maragliano and other investigators made an attempt to immunize these animals and to obtain from them a curative serum. v. Behring systematically carried out his immunization theory which he designates as Jennerization not only in a few animals but also in entire herds of cattle. According to his experience, it seems to be possible to introduce antibodies into nurslings and small children by ordinary nutrition with the milk of cows that have been rendered immune to tuberculosis, and thus to tide them over the dangerous period of contamination.

Many attempts have been made to find a specific curative serum for tubereulosis. All hopes based upon remedies new or old, or upon patent medieines, have been blasted, and the enormous amount of energy and time consumed to find a speeifieally acting serum has led to no satisfaetory results. Preventive inoculations with attenuated tubereulous virus and injeetions into the tubereulously diseased body of other bacteria antagonistie to tuberele baeilli have not found their way into practice.

In the year 1890, at the International Medieal Congress in Berlin, the eommunieation of R. Koeh that he had obtained a produet from tuberele baeilli whieh would have a specifie effeet upon the tuberculously diseased organism, this substanee being first designated as Koch's *lymph* and later as *tuberculin*, made a great furore, and awakened intense enthusiasm; but here popular enthusiasm was rapidly followed by a depressing disillusionment whieh lasted until, at the beginning of this eentury and after years of eritieal labor and study, it was demonstrated that Robert Koeh's remedy forms the basis of an etiologie speeifie therapy of tubereulosis, and that among numerous tubereulosis speeifies whieh had in the meantime been produeed his tuberculin deserves preferenee as a diagnostic remedy.

From his researehes R. Koch assumed that *incipient phthisis* eould certainly be eured by his remedy; "this may still be true of the eases not too far advaneed." It is therefore of the utmost importance for the suecess of the speeifie treatment that we use Koeh's tuberculin *as early as possible.* In doubtful eases which appear to be tubereulosis we now employ the tuberculin injections in order to make the diagnosis eertain; henee, those eases are partieularly suitable for tubereulin treatment in whieh a positive reaetion follows the diagnostic injection. R. Koch has produced various tuberculin substances; but only two of these are employed, the old tubereulin, the original Koeh's lymph, and the new tuberculin which is an emulsion of tuberele baeilli. For diagnostic purposes the former is preferable; but for therapeutic purposes after the old tuberculin has given a positive reaction in a diagnostic test, and when this therapeutie use does not produce the desired effeet, the new tuberculin should also be tried.

The teehnic is simple and presupposes strict asepsis; prior to the diagnostic injeetion, the temperature of the patient should be determined by a temperature record taken every three hours for two or three days. The Höehst tuberculin is used in a pipette divided into a hundred parts; it is diluted with boiled water in a proportion of 1 : 1,000 or 0.1 : 100, so that each division of a Pravaz syringe will then contain .1 of a milligram of tubereulin. This dose is injeeted into the back between the shoulder blade and the vertebral eolumn, and the temperature is taken early in the morning as there is usually a reaction after twelve hours. "A rise in temperature of .9° F. above the normal temperature is regarded as a reaetion. If with .1 of a milligram there is no reaction but a rise

in temperature of about .5° to .7° F., in the next injection, which should follow in about three days, a dose of .2 of a milligram will produce a distinct reaction. If, after the first injection, there is no rise in temperature, for the second injection I give .5 of a milligram, for the third, one milligram, etc., until in adults 10 milligrams at most are given; in children, according to Koch, 5 milligrams must be regarded as a maximum dose. If these doses fail to produce a rise in temperature of at least .9° F. the reaction may be regarded as negative" (Moeller).[1]

The therapeutic employment of tuberculin is the same as the diagnostic. The dose is regulated by the reaction in a concrete case, a marked individual reaction is avoided, and the injections should not too rapidly succeed each other, being usually given not oftener than twice a week. A decrease in the intensity of the reaction after large individual doses of tuberculin indicates improvement. Recovery may be assumed if, after months or years, repeated diagnostic injections of tuberculin no longer produce a reaction.

In addition to the specific treatment of tuberculosis, *symptomatic treatment* of the bowel disturbances in intestinal tuberculosis should not be left out of consideration. In far advanced cases which are unsuitable for specific treatment, or in which there are complications, the greatest weight is to be attached to the symptomatic treatment of the intestinal affection, and this must be pursued according to the plan proposed in the previous chapters.

Therefore in the chronic diarrhea of intestinal tuberculosis flushing of the intestine from above by drinking mild mineral waters as warm as possible or infusions of peppermint and other aromatic drugs early in the morning on an empty stomach, or the persistent irrigation of the intestine from below with dilute saline solutions, mineral waters (Ems, Selters, Carlsbad, Vichy), or chamomile tea may be resorted to, possibly with disinfecting or deodorizing substances, such as salicylic acid (1 : 1,000), creosote, and perhaps menthol. When there is immoderate intestinal decomposition menthol in the form of a coated pill may be given frequently and at regular intervals during the day until the odor is no longer perceptible. Thymol, which markedly corrodes the mucous membrane of the mouth, must be given in gelatin capsules, or, like salol and iodoform, in Sahli's glutoid capsules. In conclusion, small doses of opium which should be invariably given at meal-time (in pill form in combination with hydrochloric acid or extr. ligni campech.) are worthy of trial, as also the occasional administration of bismuth and tannic acid preparations and alum whey.

When there is constipation, either occasional or persistent, laxatives

[1] See chapter by Moeller upon "The Specific Treatment of Chronic Pulmonary Tuberculosis" in the "Handbuch der Therapie," etc., by Schroeder and Blumenfeld. Barth's Verlag, Leipzig, 1904.

by mouth are to be avoided, as these are liable to damage the general nutrition by wasting unabsorbed nutritive material, and the bowels must be regulated by systematic oil treatment. Occasionally, instead of pure poppy-seed oil or sesame oil, the addition of creosote to the oil ($\frac{1}{2}$ to 1 per cent.) appears to be beneficial. In inflammatory and ulcerative processes in the colon and in the rectum, oil enemata of proper proportions, regularly given at a suitable time, render excellent service. They exert a powerful effect upon the motor and sensory irritative phenomena, and favor and promote the healing of wounds by the protection which they afford to the exposed and granulating tissues. When there is marked tenesmus, an addition of tincture of opium (5 to 10 drops) to an oil enema is useful, provided the opium does not lead to constipation and the inspissation of the fæces.

With the careful observance of all the conditions which may arise, we undoubtedly have in the oil treatment a prophylactic as well as curative measure for hemorrhages and intestinal ulcers, at least the deep-seated ones, and suited to the individual requirements. By the addition of bismuth subnitrate to the oil (rubbing the bismuth with lukewarm oil in a mortar), I believe that in many cases I have promoted and hastened the healing of ulcers in the rectum and the lower colon. The effect of the bismuth and oil upon hemorrhages did not appear to be any greater than that of pure oil.

On the other hand, bismuth is a valuable remedy, if applied in substance directly to the ulcerated granulating surfaces in the rectum. This can be accomplished in the following manner: After introducing a cylindrical intestinal speculum as high as possible (and removing the obturator) the intestine is cleansed by irrigation; then by means of an insufflator, or by a glass spoon with a long handle, or by an applicator which can be introduced through the intestinal speculum, the latter being then slowly withdrawn, bismuth can be applied to the diseased intestinal mucous membrane. Touching the ulcerated surface with a 1, 2 or 3 per cent. silver nitrate solution prior to the application of bismuth has often been found beneficial, but the silver nitrate frequently so stimulated peristalsis that the bismuth was soon ejected. The same is true of liquor ferri sesquichlorati applied to bleeding ulcers and of tampons in the rectum. For this reason I have, as a rule, used locally either oil or bismuth alone, or bismuth suspended in water as in the treatment of gastric ulcer. This was injected into the intestine, and if hemorrhage from the bowel followed I administered alum whey to the patient.

In a few instances adrenalin was employed as an addition to small enemata of oil (20 to 30 drops of a 1–1,000 alcoholic solution) or as an addition to injections of water for rectal ulcers which showed a tendency to hemorrhage; I have as yet come to no conclusion regarding the value of this remedy.

In cases of ulcerating intestinal tuberculosis with profuse diarrhea and an admixture of blood in the diarrheic feces, besides such measures as are necessary to arrest the diarrhea I have administered gelatin by mouth and have also given it in small enemata. Bismuth continues much longer in suspension in gelatin than in water, therefore gelatin and bismuth enemata are sometimes very effective in the local treatment of ulcers. Finally, when tuberculous ulcers in the rectum may be reached, cauterization (under anesthesia) with a Paquelin cautery may be employed to quiet local hemorrhage. I have known this method of treatment for hemorrhages to be very beneficial. Cauterization is unsuitable for the general treatment of rectal ulcers because a single scarification is insufficient, and repetitions of the process, which are only possible under anesthesia, are too painful, the pain often lasting for several days afterward.

In the *surgical treatment* of intestinal tuberculosis many measures may be indicated, but these vary according to the individual circumstances of the case. In stenosis of the intestine in which a tuberculous process so lessens its permeability that a condition of motor insufficiency develops, the obstruction must be relieved by surgical aid or removed. In some cases the mode of operation can only be decided upon after the abdominal cavity is opened and we have recognized the exact anatomical relations by palpation with a finger or by inspection. If, in a given case of intestinal stenosis, the pathologic focus shows local limitation, resection of the stenosed intestinal area must be attempted, and if in another instance the pathologic changes show great local extension, enteroanastomosis must be performed to overcome the narrowed or occluded intestinal coils. In cases in which none of these methods of operation can be carried out because of technical difficulty, or on account of advancing debility of the patient, we must be content with enterostomy at a suitable point. In some cases of tubercular intestinal occlusion the formation of such an artificial anus is an *indicatio vitalis*. The arrest of the difficulties, and the marked improvement in local and general conditions after this comparatively slight symptomatic operation justifies its performance also in cases of intestinal tuberculosis in which there is no organic stenosis, but in which, however, extensive ulcerative processes in the colon cause profound subjective disturbances, such as severe diarrhea which cannot be controlled by internal remedies, and especially hemorrhage. Colostomy above the diseased intestinal region excludes the latter from taking part in the labor of digestion, induces much better conditions for the healing of the ulcer, renders possible a more active local treatment of the diseased intestinal mucous membrane, and, what is more important, enables us to give sufficient nourishment and a general treatment. Whether the artificial anus must later be closed depends upon the further progress of the case.

Tuberculosis of the rectum most frequently necessitates surgical meas-

ures because inflammatory and ulcerative processes in this portion of the intestine which is not covered by peritoneum readily penetrate from the intestinal wall to the perirectal cellular tissue, or perforate it and there form abscesses which must be incised. Both incised and spontaneously perforating perirectal abscesses leave the familiar internal or external anal fistula which may also be complete, for the treatment of which extensive division through the sphincter is the best remedy.

The tuberculous patient in all stages of his disease needs every possible advantage that can accrue from a hygienic mode of life and careful nursing. Patients with intestinal stenosis do not reap the same benefit from treatment in sanatoria and large institutions for the cure of tuberculosis as those suffering from pulmonary tuberculosis, since, owing to the nature of the intestinal disturbance, their nutrition necessitates greater specialization than is practicable in a large institution in which general over-nutrition is to be carried on for some time. Nor are they adapted for treatment in high altitudes and at the seashore, for they are often very susceptible to changes of temperature; hence bath cures, salt baths, and (when intestinal tuberculosis is combined with peritonitis) peat baths can only come into consideration after the intestinal disturbance shows a certain degree of improvement or has become latent.

Until such amelioration occurs, *nutritive therapy* must be one of the chief aims of the treatment, and for this the fundamental laws have been detailed in preceding chapters. As soon as the condition of the intestine will warrant it we order a mixed diet, preferably of boiled flour foods, in such quantities as may substitute for losses which have taken place in consequence of fever, diarrhea and the under-nutrition due to the latter, and will finally bring about an increase in weight. Care and observation by coproscopy will determine the proper additions of fat to the nutrition, for it has been proven by experience that in intestinal dyspepsia due to tuberculosis fat absorption suffers early and continuously, and in such a way that even the most perfect of foods, milk, is often not well borne.

CONSTIPATION AND HEMORRHOIDS

By J. BOAS, Berlin

Constipation and hemorrhoids, as is well known, belong to the most common affections of the intestinal canal. Perhaps because scientifically resultless, some physicians regard these conditions as a *quantité negligeable* —for there is in medicine a certain unmistakable timocratic principle. Possibly, even to-day, the opinion is extant that constipation and hemorrhoids are slight, though troublesome, evils, and, here and there, an impression exists that internal medicine taken altogether is powerless permanently to relieve this condition.

I hope to convince all that neither of these views is correct. Above all, I wish to state explicitly that the treatment of constipation and hemorrhoids is one of the most fruitful fields of internal medicine, and that it is our imperative duty as physicians thoroughly to acquaint ourselves with this condition since it represents the principal branch of the so-called "Nature Cure." It would be actually derogatory to our science if we were unable to obtain similar, if not better results than these empirics.

But, before proceeding to the treatment of habitual constipation and hemorrhoids, I shall first define and portray our conception of the first mentioned symptom-complex.

CONSTIPATION

We may differentiate between physiologic and pathologic constipation. Constipation is physiologic so long as the delay in the evacuation of feces produces no disturbance, not even slight, in the intestinal tract or in remote organs. These physiologic effects we have learned to recognize in other organs, for example, in the heart (physiologic bradycardia), in the female genitalia (appearance of menstruation every two or three months), etc.

Within these limits the degree of diminished activity may vary widely. Much found in earlier literature, for example, the report that patients have had only one or two evacuations in a year, may be referred to the domain of legend. But I have certainly seen a patient who had a fecal evacuation only once in four weeks, and then it had to be each time extracted digitally by a nurse.

This patient, on account of some gynecologic affection, had undergone several laparotomies; probably adhesions had formed between different por-

tions of the intestines. In every other respect, however, her condition left nothing to be desired. Her appearance was robust, the urine was normal, and the amount of indican, in particular, was not increased.

In another case, that of a lady, aged 69, I obtained the history that the patient had been small and weak from her youth. Only once a week was there any desire to have a fecal evacuation. She had experienced no discomfort from this throughout her entire life, but she later succumbed to carcinoma of the esophagus.

Nevertheless, such diminished physiologic activity must, at least, be looked upon as a chronic anomaly and is certainly rare. Usually this results in a slow or rapid systemic disturbance, and physiologic constipation leads to true *pathologic* habitual constipation.

But we must be even more precise than this:

Not every case of chronic constipation can be designated as habitual. On the contrary, by custom this nomenclature has been applied only to those anomalies in which, in the first place, there are no other primary disturbances; and secondly, to the cases in which the intestinal evacuation as such is nowise due to mechanical or toxic effects (for example, lead colic). It is readily seen that such a limitation cannot possibly be absolute, that under the picture of simple habitual constipation all sorts of pathologic pictures, occasionally serious and prognostically unfavorable ones, may be masked. Nevertheless, we must be content, as elsewhere in the practice of medicine, with a symptomatic diagnosis so long as we have no deeper insight, so long as other decisive factors have not been added, or, finally, while the entire course of the disease receives another interpretation.

Here, at the beginning of our description, we must necessarily be suspicious of the diagnosis of habitual constipation in those cases in which the facts are not absolutely uniform and clear. In the chapter devoted to diagnosis we shall have an opportunity to justify this position *in extenso*.

ETIOLOGY

ETIOLOGY OF HABITUAL CONSTIPATION

In consonance with the above definition we can only enumerate those causes of habitual constipation in which organic changes, no matter of what nature, are absent. The number of secondary factors which induce habitual constipation is so great that an exhaustive compilation is impossible.

The causal factors of idiopathic constipation are practically important because with their removal the affection is occasionally cured.

In unbiased judgment, we must exclude a large group in which a distinct, causal etiology cannot be recognized. The affection is mild at the

onset, but the intestine becomes sluggish, although the patient attaches but little importance to this until gradually the necessity for relief causes a resort, usually to purgatives, more rarely to injections. As the former gradually lose their effect, more active purgatives are employed, and the condition is thus carelessly allowed to continue year in, year out, until, finally, all remedies are inactive, or pain and catarrhal phenomena necessitate rational and comprehensive interference.

Habitual constipation may frequently be referred to *hereditary influences.* Constipation to a greater or less extent prevails in entire families. Occasionally the condition has its beginning, as I have repeatedly observed, in the first days or weeks after birth, and even in this early stage it is sometimes very difficult to combat.

This hereditary predisposition is often combined with a lack of true "*bowel training.*" The children are not taught to observe regularity in regard to their bowel movements or—and this is particularly the case with young girls—false modesty leads the children to artificial suppression. This sin is most frequently committed in schools, while upon visits, and in summer resorts.

In rare cases this form of constipation may be due to a dilatation of the colon (probably congenital), the so-called Hirschsprung's disease, the prognosis of which, even under the operation recently proposed, is very unfavorable.

A frequent cause of habitual constipation, which I have called *alimentary constipation,* is due to improper nutrition; in particular, the immoderate ingestion of meat and the avoidance of vegetables leaving a residue, as well as the use of rye bread and varieties of sugar.

The physician himself is responsible for *alimentary constipation* more frequently than is generally supposed. As an example, I may mention the diet prescribed for diabetes, which, as employed in most cases, necessarily produces constipation. A milk diet, too, as ordered in anemia, chlorosis, and nephritis, frequently has an unfavorable effect, or, at least, it does not produce the desired result because the constipation which usually follows the use of a milk diet is too little considered.

To this must be added another common form of constipation which is not sufficiently borne in mind, and, indeed, is not generally recognized: *That due to the employment of drugs.* In acute intestinal disturbances and in cholera nostras, but particularly in appendicitis, and probably also in other forms of peritonitis, in occlusion of the bowel or in pseudo-ileus, large doses of opiates or the belladonna preparations are frequently used or, more correctly, wasted, and this is followed, as I have seen in innumerable instances, by severe and most unyielding intestinal paresis. Besides opiates, substances containing tannic acid are often too long employed for diarrhea, and, occasionally even after a short time, produce constipation; in the course of years I have seen several well developed cases of this

kind. We can scarcely be wrong in assuming that extremely severe changes in function are due to an abnormal tannic acid effect.

As a curious abnormality, I shall here call attention to a natural increase of habitual constipation which I have repeatedly observed in my practice: The purposely limited ingestion of food. While it does not seem plausible, many individuals, mostly neurasthenics, have an idea that by a natural limitation of the ingestion of food they may relieve their difficulties due to constipation.

A much more frequent cause, and one which requires less consideration than the foregoing, is the familiar one, *lack of exercise*, which has been much discussed, and which also plays an important rôle in the etiology of hemorrhoids, as we shall later have occasion to note. The influence of active exercise and, where this is impossible, of passive exercise cannot be doubted, yet we must be careful not to exaggerate the importance of this as a causative factor. For example, it is generally known that extreme habitual constipation is found in persons whose occupations certainly indicate no want of exercise, for example, in farmers, army officers, gymnasts, bicyclists and others. On the other hand, I have several times called attention to the fact that in certain forms of habitual constipation which will shortly be described bodily rest is much more likely to relieve the affection than forced exercise.

One of the chief causes of habitual constipation is *neurogenous*, or *psychogenous*, which is a better term. It is an almost constant accompaniment of general neurasthenia, but may occasionally present itself as a substantive intestinal neurosis.

A widely distributed type of neurogenous constipation is found in enteroptosis, the relation of which to neurasthenia cannot to-day be doubted even though their intimate connection is still a mooted question. As acute psychical factors may generate a state of anxiety, an irritation of peristalsis from nerve tracts which we refer to the splanchnic area, so by chronic irritation there may be a permanent diminution of the energy of innervation.

I recently treated Miss D., a teacher, aged 34, who, since Whitsuntide, 1901, had suffered from occasional diarrhea, swelling of the feet, pain in the left hypochondrium, insomnia and great lassitude. During her holidays there was marked improvement, but soon after resuming teaching marked nervousness and constipation simultaneously appeared. Purgatives, to which the patient, however, had never accustomed herself, were without effect. In the beginning simple enemata, and, later, even enemata of oil, were ineffectual. She ceased to teach, and the condition of the bowels steadily improved until gradually there was a profuse and spontaneous evacuation daily. Constipation reappeared only after excitement, even though of a pleasant nature.

In other cases of psychogenous constipation, the greatest variations between constipation and diarrhea may be observed.

[As an additional cause of chronic constipation, one finds in America

the habit of irregularity and postponement of defecation because of the discomfort of the American outdoor privy, especially in the winter months.

It is to be noted, too, that child-bearing women who suffer from diastasis of the recti abdominalis muscles and consequent weakness of the abdominal walls suffer more or less from ptosis of the intestines. The weakened abdominal walls prevent, too, the abdominal press, which is so necessary in the act of defecation.

Anal fissure and other painful conditions of the fundament with resulting spasm of the external sphincter result in constipation, chiefly because of the fear of pain which a bowel movement produces. Sphincter spasm is also a direct cause of constipation.—ED.]

ETIOLOGY OF HEMORRHOIDS

Any of the causes which lead to habitual constipation will soon or late cause a more or less marked development of hemorrhoids. Stasis of the intestinal contents in the lowest portions of the intestine from the sigmoid flexure downward is a particularly active factor; there is congestion of the hemorrhoidal veins which no longer have power to empty their blood normally into the inferior vena cava. Other causative factors are also operative, but they are alike in the fact that, externally or internally, they inhibit propulsion to the great reservoirs of the abdomen. The most important are: Tumors of the rectum, hypertrophy of the prostate glands, uterine retroflexion, ovarian tumors, hematocele retrauterina, myomata and fibromata of the uterus, various affections of the bladder, and also large tumors of the pelvis, etc.

Hemorrhoids form a very frequent and distressing complication in *pregnancy*. As a rare etiologic factor, cholelithiasis has been mentioned; this, however, has little in common with hemorrhoids except that, in this condition, habitual constipation is exceedingly common. A hereditary predisposition to hemorrhoids has already been spoken of, but here, probably, the factor of hereditary constipation which so invariably occurs must be taken into consideration.

Although constipation and hemorrhoids show an unmistakable symbiosis, other less frequent causes must not be undervalued. Thus, the decided formation of varices unquestionably occurs also in chronic diarrhea, as may be readily understood when their seat and cause are in the lower portions of the intestine. In the course of years I have observed quite a number of such postdiarrheic hemorrhoidal formations.

In recent text-books circulatory disturbances are frequently cited as the cause of varices. As, however, Esmarch [1] and Nothnagel [2] quite prop-

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[1] *Esmarch*, "Krankheiten des Mastdarms," p. 168.

[2] *Nothnagel*, "Darmkrankheiten," 2 Aufl., p. 469.

erly maintain, the pathologic increase in pressure, even with severe cardiac insufficiency, distributes itself over such an extensive vascular area that it can scarcely be operative in the hemorrhoidal plexus in the manner supposed.

In conclusion, age and sex must be enumerated among the causal factors. Varices in earliest childhood are extremely rare; their occurrence in later years reveals a marked difference in regard to sex, inasmuch as the male sex shows proportionately a much greater liability than the female sex, while the inverse is true of habitual constipation. In my opinion, the explanation of this can only be found in the more numerous venus plexuses of the female genitalia, the collateral circulation of which furnishes an outlet for the stasis of blood.

SYMPTOMATOLOGY

SYMPTOMS OF HABITUAL CONSTIPATION

In the simplest and mildest form of habitual constipation the symptoms consist in the incapacity of the intestine to expel the intestinal contents within twenty-four or forty-eight hours. But, with accurate observation, we may discuss somewhat more minutely the degree and nature of diminished intestinal peristalsis.

(a) In the large intestine, *in toto,* particularly, however, in its upper portion, muscular activity is lessened.

(b) The intestinal contents cannot be propelled normally to the lower portions of the intestine, congestion occurring in the sigmoid flexure and in the rectum. These portions then become the seat of a deficient power of expulsion.

In both cases the ultimate effect is the same, but scientifically, and, as we shall see later, also therapeutically, the point at which the deficient power of expulsion exists is more or less essential.

According to recent experiences (Fleiner, Westphalen and others) and clinical observation, we may differentiate four varieties of habitual constipation: First, the *atonic spastic form.* It must be expressly stated that the differentiation between atonic and spastic constipation is somewhat artificial. In the former, according to Fleiner, the bowel contents are said to be more compact and drier than usual; they consist of clumps closely adherent or of cylindrical masses of large caliber, sometimes even of individual particles or scybalæ bearing a visible impression made by the haustra of the intestine. Secondly, the spastic form is said to consist of small, friable, pediculated cylinders, usually of about the thickness of a lead-pencil, or of globular fecal masses the size of a hazelnut. Since, however, as Fleiner has shown, numerous transformations and combinations of these forms take place—any one can observe this personally at any time—Fleiner's classification is of little consequence. More distinctive than the

formation of the stool is the proof of spastically contracting intestinal coils, which we shall consider later. In my experience these conditions vary greatly in the same individual. Nevertheless, continuous and extensive spastic contractions are frequently found associated with a spastically formed bowel movement.[1]

As a third form of habitual constipation that variety characterized by me as a *fragmentary bowel movement* must be differentiated. In this condition, as in the case of hemorrhoids, proctitis, or rectal carcinoma, there is marked tenesmus which compels the patient to seek the toilet every few hours, yet the abdominal pressure brings forth only a few particles of feces. I have usually observed this condition in patients suffering from intestinal neurasthenia.

Finally, we must mention a fourth form which is distinctly characterized and very frequent: *Stercoral diarrhea.* In this affection there is at first constipation which lasts from one to three days, sometimes even longer, and is accompanied or followed by severe colicky intestinal pain and rumbling, and by one or several copious diarrheic discharges; these are at first firm, then fluid, but may be diarrheic from the start. The same typical symptom-complex then reappears, varying only in severity, frequency and duration, both in the attacks and the intervals.

These are the most common and comparatively the most typical forms of habitual constipation. Besides those mentioned there are undoubtedly other modifications which cannot be described.

More necessary is the description of two important and, occasionally, very distressing and painful symptoms of constipation: *Flatulency* and *intestinal colic.* The former is either due to abnormal filling of the intestine with putrid gases which the intestinal musculature is powerless to expel, or it may be attributed to an abnormal and excessive production of gaseous products while the intestinal motility is good. In the former case, therefore, gas production is normal but there is diminished intestinal peristalsis; in the latter case there is an abnormal and copious gas formation with sufficient intestinal motility. In the last, the bowel movements may be sufficient and regular.

Without doubt the first mentioned form is by far the most unpleasant, and is intimately connected with the colic now to be described. But the second form, also, is no less unpleasant to the patient than to those about him. Intestinal colic, as mentioned, may be due either to abnormal retention of gas (flatulent colic) or to other non-gaseous products of decomposition (stercoral colic). In the former case the attack of pain after

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[1] However, spastic intestinal contractions are by no means always pathologic. I have seen them also when the composition of the feces was quite normal. In chronic diarrhea (not stercoral diarrhea) I also demonstrated well-developed, contracted, intestinal coils.

more or less time ceases with the passage of gas; in the latter, euphoria only follows a copious fecal discharge.

From this true intestinal colic we must carefully differentiate certain gastric difficulties to which Knud Faber [1] has recently called attention, and whose experiences we can absolutely confirm.

Another condition which belongs to the symptom picture of habitual constipation has, since Bouchard's celebrated researches, been designated as *autointoxication*. Headache, sometimes appearing as pressure in the cranium and sometimes as migraine, also dizziness and lassitude here play an important rôle. In fact, we cannot at once reject the theory of a causal connection here, and every physician of experience will recall many cases in which the above mentioned symptoms, particularly if not well developed, permanently disappeared on regulating the bowels.

On the other hand, it would be a manifest exaggeration to attribute to migraine all attacks resembling migraine and running their course with habitual constipation. Only too frequently it becomes my duty to dispel the illusions of my patients who suffer from migraine and refer their condition to faulty gastrointestinal digestion, and the fruitlessness of treatment directed to this condition has almost invariably shown me to be correct.

Finally, the well-marked picture of neurasthenia is frequently combined with habitual constipation, as has already been mentioned, and in these cases, objectively speaking, the neurasthenia cannot be regarded as the consequence but the cause of chronic intestinal inactivity.

Habitual constipation may also exert an influence upon the *kidneys*; thus, as Kobler [2] and Ebstein [3] have observed, and as I may confirm, occasionally slight albuminuria and cylindruria are met with. In how far we are here dealing with a mild grade of nephritis or a true coprogenous albuminuria cannot at present be determined. Kobler's investigations favor the view of coprogenous albuminuria. According to my own experience, albuminuria and cylindruria undoubtedly form the rarest complications of habitual constipation.

SYMPTOMS OF HEMORRHOIDS

Hemorrhoids, on account of their intimate connection with habitual constipation, naturally present many of the same symptoms. But the localization of the affection and the formation of varices add to the previously described picture a few peculiarities.

The explanation of these is found in the mechanical effect which they produce. As such, the atony in the lowest portion of the large intestine

[1] *Knud Faber, Arch. f. Verdauungskrankheiten*, 1902, Bd. VIII, Heft 1.

[2] *Kobler, Wiener klin. Wochenschrift*, 1898, Nr. 20.

[3] *Ebstein*, "Die chronische Stuhlverstopfung in der Theorie und Praxis." Stuttgart, 1901, p. 86.

and in the rectum must be first mentioned. This causes stagnation of the intestinal contents in the sigmoid flexure and in the ampulla recti, and also the development of a catarrh of the rectum, occasionally even of the sigmoid flexure. Tenesmus, which is very frequent, is also due to stagnation of the intestinal contents in the rectum and in the sigmoid flexure.

Occasionally the propulsion of the contents becomes difficult from a purely mechanical cause, namely, the extension of the varices and nodules. Thus a vicious circle is formed, for the varices increase the constipation and, *vice versa*, the latter condition aggravates the hemorrhoidal affection.

Another source of difficulty is the ready tendency to inflammation or even ulceration of the nodules (which have extraordinarily thin walls) from the continuous mechanical irritation to which they are exposed, this being favored by a lack of cleanliness. This gives rise to the itching and burning of which the patients so frequently complain, and occasionally also to severe, boring, unbearable pain combined with the most distressing tenesmus. Not infrequently the pain and burning sensation are felt not at the point of affection but higher up, in the lumbar region or in the shoulders, and quite typical attacks of sciatica may occur—I have seen several undoubted cases of this kind—which, if the origin of the trouble is not located, may lead to various therapeutic errors.

The most common manifestation of hemorrhoids, as is known to every layman, is the periodically appearing *hemorrhages*. These may vary in degree and frequency. It was formerly assumed that the hemorrhage from hemorrhoids was beneficial, and to some extent a self-regulation of the condition of stasis in the portal vein circulation.

Upon unprejudiced testing we must admit that there is at least a grain of truth in this view, inasmuch as, after diffuse depletion, there is a transitory decrease in the varices distended with blood and, hand in hand with this, euphoria of shorter or longer duration may appear. Beyond this there is no salutary influence from the bleeding of hemorrhoids; for, in the first place, the expected amelioration from loss of blood does not always follow, and, moreover, from their duration and intensity, the latter may produce severe anemia without in the slightest degree diminishing the local affection. The proverb, "All that glitters is not gold," is also true of "the golden vein." The formation of hemorrhoids is intimately connected with enlargement of the liver. I cannot express this relation in figures, but all who are accustomed carefully to percuss and palpate the liver of patients with hemorrhoids must have noted the frequency of hepatic congestion.

One of the most positive and interesting cases of this combination is the following: A hotel proprietor, aged 40, but an exceptional example of temperature in regard to alcohol, for many years had been suffering from habitual constipation and highly developed external and internal

hemorrhoids. The liver was four fingerbreadths below the margin of the ribs; there was no enlargement of the spleen. The appetite was good, the patient digested all food without difficulty. From time to time the enlargement of the liver increased decidedly, and caused a feeling of pressure and tension in the abdomen. Whenever hemorrhage from the hemorrhoids appeared, the liver decreased in size, and the symptoms disappeared. Instead of bleeding from the hemorrhoids sometimes there was very copious hematemesis; no symptoms of ulcer or gastritis were ever observed, but in the course of time there were six severe attacks of hematemesis. Without forcing scepticism too far, we are here actually dealing with a case of *vicarious gastric hemorrhage.* This, therefore, is not, as v. Schrötter [1] has recently maintained, a canard (Räubergeschichte).

Earlier literature, which found fanatic expression chiefly in the teachings of Georg Ernst Stahl, ascribed to hemorrhoids a far greater importance than that just alluded to. According to this, the stoppage of hemorrhoidal bleeding was said to produce all sorts of internal diseases (gout, diseases of the liver, the heart, the lungs, and others). There can be no doubt that this teaching over-reached its mark, and was justified only in so far as congestion in the deeper areas, occasionally also in organs situated higher up, was affected by the portal vein circulation.

DIAGNOSIS

DIAGNOSIS OF HABITUAL CONSTIPATION

In turning to the diagnosis of habitual constipation and hemorrhoids, the recognition of these affections at first sight appears very simple, and to require no special elucidation. In fact, this is true in the overwhelming majority of cases. But even this apparently simple clinical picture may cause perplexity in diagnosis, and it is therefore absolutely necessary that patients complaining of *habitual constipation* be examined just as thoroughly as those suffering from any other affection.

The important question to be decided in every case is this: Is the constipation habitual, viz., primary, like that previously described, or is another causal factor present? At the beginning of this article we called attention to the numerous causes which may produce this condition; possibly their rôle is not always important, but a thorough diagnostic insight is also a valuable aid in the treatment.

At this point we shall busy ourselves especially with the various diagnostic difficulties which are referable to the digestive apparatus itself.

It is a common experience to find that patients with various disturbances of digestion regard habitual constipation as the most prominent and

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[1] *v. Schrötter, Verhandl. des Congresses für innere Medicin,* 1902. "Discussion zu dem Vortrage von Ewald, Ueber die Diagnose des Magengeschwürs."

frequent cause of these, which occasionally, however, masks other important symptoms. Thus, I have frequently noted that patients with stenosis of the pylorus, even with gastric carcinoma, complain merely of habitual constipation, while the serious underlying gastric affection is revealed only by a careful examination.

The possibility of deeply situated intestinal disease is more significant. First in importance is carcinoma of the intestine, especially that involving the colon, not on account of its frequency, but because it may lead to grave errors. Of the entire structure of the symptom-complex only constipation can be discerned upon careful, and especially upon superficial, examination. What is true of carcinoma is also true of benign stenosis. A less serious error, but nevertheless not without interest, consists in the non-recognition of the combination of habitual constipation with colitis, to which we shall refer later.

These considerations, which by no means exhaust the differential diagnosis, sufficiently indicate that the diagnosis of habitual constipation must, under all circumstances, be preceded by a thorough local and general examination.

The special diagnosis which then follows must comprehend the physical investigation of the intestines and the feces.

In the former, the recognition of intestinal atony by filling the bowel with water or gas is easy. The same condition is pointed out by the finding of contracted intestinal coils, especially of the transverse colon, the cecum and the sigmoid flexure, which also indicate the spastic character of the chronic constipation. Extreme meteorism favors stagnation of the contents; on the other hand, sensitiveness to pressure in the region of the colon is not common in simple, habitual constipation, but indicates colitis.

The examination of the rectum is an integral part of an examination of the intestinal canal. Aside from the discovery of hemorrhoids, which will be fully described later, ampullar bulgings will probably be observed which indicate stagnation of the dejecta in the lower portion of the bowel. If large scybala are recognized in the rectum, the correct remedy may be at once employed.

In conclusion, observation of the *bowel movement itself* is a valuable aid in the recognition of the nature and the extent of the constipation. Particles of mucus should be examined, provided a so-called *exploratory irrigation of the intestine* is not undertaken; these I always advise, particularly if the patient reports the appearance of mucus, or if there is sensitiveness to pressure over the colon.

For the physician not familiar with intestinal diseases it may here be remarked that irrigation of the intestine is carried out in a manner similar to gastric lavage. For this purpose a soft rectal tube is attached by means of a small glass tube to a rubber tube about a yard and a half

long which has inserted, at its upper end, a glass funnel. The funnel is elevated and water at a temperature of about 30° C. is permitted to flow into the intestine; upon lowering the funnel the fluid is returned and received in a vessel held for this purpose. This fluid may then be readily examined for mucus and other pathologic constituents.

DIAGNOSIS OF HEMORRHOIDS

The *diagnosis of hemorrhoids* necessitates a local inspection of the anus, and a *digital examination* of the rectum. This examination, which is really self-evident, is unfortunately, notwithstanding the urgent advice given in text-books and medical journals, not sufficiently often made. The operative treatment of rectal carcinoma, which is too often unrecognized and sails under the flag of hemorrhoids, would then give us much more favorable results.

In every case of hemorrhoids it must also be determined whether we are dealing with *external, internal,* or *mixed* hemorrhoids. While the first are readily recognizable, the diagnosis of internal hemorrhoids may occasion great perplexity. Internal hemorrhoids are most readily revealed by strong pressure, and best after injections of a hot saline solution or glycerin.

Where nodules do not thus appear, and the bleeding from the anus indicates a hemorrhoidal origin, it is possible, as J. Schreiber [1] has lately shown, to determine the source of the hemorrhage by the rectoscope, and to control and treat it surgically. Similar cases have been described by Nothnagel [2] and Ewald.[3] According to Schreiber, hemorrhoidal venous dilatation is observed much higher in the intestine than was previously thought possible, even as high as the sigmoid flexure.

The differential diagnosis and the complications of hemorrhoids require a few brief remarks. In regard to the former, it must be emphasized that the confusion of hemorrhoids with other diseases of the rectum, provided the rectum is carefully palpated, is very rare. At most small polypi or angiomata may cause perplexity. The differentiation of a beginning carcinoma from ulcerating hemorrhoids is sometimes difficult, all the more so since from these cancroids may develop in the course of time. Successive palpations and rectoscopic examinations, perhaps the excision of a portion under narcosis or local anesthesia, will in most cases clear the situation.

The *complications* of hemorrhoids are numerous. Catarrh of the rectum (proctitis haemorrhoidalis) has already been mentioned; this fre-

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[1] *J. Schreiber*, "Die Rectoromanoskopie." Berlin, 1903. p. 73 et seq.

[2] *Nothnagel*, "Zur Klinik der Darmkrankheiten." *Festschrift z. Feier d. 60. Geburtstages von Max Jaffé.* Braunschweig, 1901.

[3] *Ewald*, "Klinik der Verdauungskrankheiten," 1902, III, pp. 425 u. 426.

quently runs its course with tenesmus. On strong pressure we observe, as a rule, the discharge of a gelatinous yellowish-white mucus.

In the course of hemorrhoids more or less deep ulceration often occurs, and causes severe burning or pain. Careful examination with the speculum will usually enable us to recognize this complication.

Occasionally there is incontinence of the sphincters of the rectum, which, even without the prolapse of nodules, leads to familiar disturbances, particularly to the discharge of thin, fluid feces. Inspection and digital examination will readily reveal this condition.

When hemorrhoids have persisted for a long time, hemorrhoidal prolapse frequently develops and may vary greatly in severity. In the milder cases prolapse only occurs upon defecation. In more advanced cases it may follow any movement, for instance, sneezing, the passage of flatus, etc.

TREATMENT

TREATMENT OF HABITUAL CONSTIPATION

Where physico-dietetics have been added to true drug treatment, various methods for relieving habitual constipation are to-day resorted to by physicians. The decision as to the efficacy of the different measures is made extremely difficult from the fact that the various curative agents employed in practice are so numerous, and are even multiplied in spas and sanatoriums. This perplexity in judging as to the effectiveness of individual drugs and methods is still further enhanced inasmuch as they are sometimes employed in mild, at other times in severe cases. Upon accurate investigation the usual criteria for this are not positive; therefore, at the beginning of this section, a brief outline of the degrees of constipation is not out of place.

Upon superficial judgment, the best standard appears to be the duration of chronic constipation. This will often, but by no means invariably, be found a true measure. I have had under observation many patients who, for twenty or thirty years, had been able to regulate their intestinal functions only by drugs, and, by the institution of a proper diet, they were at once completely and permanently relieved. Inversely, we sometimes see patients in whom constipation has existed but a short time, that is, for one or two years, yet, contrary to all expectation, curative measures are absolutely ineffectual.

More exact tests, but by no means always decisive of the degree of habitual constipation, are the effect and the dose of the remedies or curative measures which have been employed. Here we may differentiate between laxatives, purgatives, and drastics, and between these are the so-called neutral salts. In some cases mere laxatives—and among these I count rhubarb, preparations of frangula, cascara sagrada, tamarinds,

compound licorice powder, flowers of sulphur and the like—in small doses are sufficient, sometimes for years; these cases are, therefore, unquestionably mild. In others these remedies are without result or are effective only in very large doses. Such cases may be designated as moderately severe. The most extreme degree of chronic constipation is represented by the patients in whom the neutral salts, and even drastics in large doses, either fail to bring relief or act insufficiently. But another group is also to be differentiated, and in these neither mild nor drastic purgatives produce free action, while the simple injection of water at once gives relief. These, apparently, are the cases in which the lower portion of the intestine is the seat of intestinal atony, and here we must bear in mind the psychogenous or neurogenous group in which purgatives, injections, and massage, etc., are without effect unless supplemented by the complete removal of the patient from his former surroundings and the cares of his occupation.

Only by such a separation can we decide as to the nature and degree of the affection, and only thus can we correctly estimate the value of the many remedies and methods employed to relieve the condition.

Before proceeding with our discussion it is by no means superfluous for us to ask: What is meant by the cure of habitual constipation? In my opinion we can only speak of habitual constipation as cured if, a year after the termination of the treatment, there is a spontaneous and ample evacuation every twenty-four to forty-eight hours.

If all physicians were to accept this as a standard, our confidence in the efficacy of measures which have apparently been very successful would soon be shattered.

As soon as we lower our standard, and regard temporary improvements as cures, the number of successful remedies is, of course, very considerable.

In the following, the treatment of habitual constipation will be considered only from that standpoint which we have established as indicating a cure.

As everywhere in medicine, *prophylaxis* plays the leading and most important rôle. It begins in childhood with the consideration of the previously mentioned congenital predisposition to constipation. In addition to this, it must be the object of home training and of our professional endeavors to instruct children as to the importance of regulating their intestinal functions. To attain this end a thorough understanding of the process of nutrition is, above all, necessary.

We know that, especially in the better classes, the desire for a strengthening diet takes precedence of all other considerations, and the mistaken parents regard expensive meat and the nutritious egg as the only nourishing, or, at least, the best food.

Aside from the fact that a too stereotyped diet fails to produce the

nutritive effect expected (to the astonishment of those who institute it), another undesirable factor is added—habitual constipation. It is therefore the duty of the physician to regulate the nutrition of children, and from time to time to add to the uniform, constipating diet food which favors the regular action of the bowels.

What is true of children also applies to the condition in adults designated by me as alimentary constipation. In particular, the table d'hôte with its abundance of meat and fish in contrast with its usually small amount of vegetables is a frequent cause of constipation in young persons.

Here, in a restricted sense, the same laws of treatment are operative which, in Germany at least, Albin Hoffmann first utilized with such marked success in therapy: *exercise* and *abstinence*. In alimentary constipation exercise is the more necessary; upon this, in fact, are based our most important methods of treatment, which range from ordinary walks to Swedish movements and massage. A practical and even a decided influence is to be ascribed to the diet inasmuch as here thermic, chemical and mechanical stimulation, occasionally all combined, stimulate peristalsis to an increased activity. *As a practical, pioneer factor, the diet, therefore, occupies a leading place among the curative agents.*

In the first place, the diet of chronic constipation, like the diet in other affections, is based upon the law of experience. To the present time the sanction of inductive science is lacking. Nevertheless, we may picture to ourselves more or less completely the effect upon the intestinal function of an increase in diet (I designate this as *constipation diet*).

It is generally known that certain foods stimulate and others inhibit intestinal activity just as do certain drugs, and upon exact investigation the line of demarcation between the effect of a drug and that of the food is almost obliterated. Whether I administer tannic acid in the form of acidum tannicum or as acorn coffee, whether bilberry wine or red wine containing a large amount of tannin, whether rhubarb compote or an infusion of the root of rhubarb, whether I give fig syrup or half a dozen fresh figs certainly makes no fundamental difference; at most, in the one case we are employing the raw substance, and in the other we are administering the active principle in as pure a form as possible.

The physiology of food gives us a large number of such remedies which stimulate the intestine. We may divide them into the following groups:

1. *Substances containing sugar.*—In this group belong common cane sugar, milk sugar, honey, manna, levulose, dextrose, sweet whipped cream, fruits containing large quantities of sugar (plums, grapes, figs, oranges, dates, etc.). The effect of sugar is probably manifold. In the first place, solutions of sugar in the stomach produce active secretion (dilution secre-

tion, Strauss), and in the sugar not at once absorbed acids of fermentation develop.

2. *Foods containing organic acids.*—Among these are: Buttermilk, sour milk, kefyr, kumiss, sour whey; also fruit wines, for example, cider or Moselle wine, currant wine, gooseberry wine and, in Russia, the so-called "kwass"; sour lemonade (made from lemons), acid fruits (apples, sour cherries, gooseberries and currants, etc.); rye bread, characterized by its amount of sour dough, belongs to this category.

3. *Salty substances.*—As the commonest remedy, ordinary table salt in its various combinations (salt water, salt herring, caviar, sardellen, etc.). Smoked and pickled foods also belong to this category.

4. *Substances containing carbonic acid or those which form carbonic acid.*—In this group belong the natural or artificial waters which contain carbonic acid, also the simple alkaline waters, and the above mentioned kefyr, kumiss, kwass, matzoon, etc. Yeast and yeast bread may also be included in this category. Finally a number of so-called gas-forming vegetables; these produce CO_2 as well as other varieties of gas.

5. *Substances which contain fat, and in which the amount of fatty acid assists the action.*—The most common remedies here are butter, olive oil, sesame oil, or linseed oil, which is generally and quite properly, I think, regarded as difficult of digestion. Perhaps the value of linseed (see later) depends upon the large amount of fat it contains. Here, on account of its contents of fat, the so-called mayonnaise is to be included, as well as Italian salad and, perhaps, all salads prepared with oil.

These are the main chemical stimulants to the intestine, and to these must be added the thermic and mechanical ones.

6. On account of its thermic effect only *cold,* naturally, must be considered. This is best employed as a drink in the form of cold water, cold milk, either sweet or sour, cold lemonade. Cold soups (such as fruit soups, and the like) have, in addition to other effects, also a certain thermic influence. On the other hand, cold or frozen fruits, because usually introduced into a full stomach where equalization of temperature soon occurs, are less beneficial. The action of these substances depends mainly upon the state of the stomach, whether full or empty, and the optimum of the thermic effect is consequently found in an empty stomach, a fact daily demonstrated in practice.

7. The *mechanical* effects of food are manifold. The more the indigestible residue from a food, *cæteris paribus,* the more it promotes the passage of the intestinal contents.

The greater the proportion of food absorbed in the intestinal canal, the less suitable is it in the treatment of habitual constipation.

We are indebted to the researches of Rubner for our knowledge of the absorption of food; if not of all at least of the most common ones. According to these the percentage of non-absorption is as follows:

	Of the dried substance	Of albumin	Of carbohydrates
In broiled and fried meat	4.9–5.3	2.0–2.6	...
In shell fish	4.3	2.5	...
Hard boiled eggs	5.2	2.6	...
Milk	8.8	7.8	...
Wheat bread from fine flour	4.2	21.8	1.1
Rolls	5.6	22.2	2.9
Rye bread from coarse ground corn	13.1	36.7	2.9
Rye bread from the whole corn	20.9	46.6	14.3
Pumpernickel	19.3	43.0	13.8
Rice	4.1	20.4	0.9
Peas	9.1	17.5	3.6
Green beans	15.0	....	...
Beans	18.3	30.2	...
Mashed potatoes	9.4	30.5	7.4
Savoy	14.4	18.5	15.4
Yellow turnips	20.7	39.0	18.2

This Table is very instructive for our purpose. It shows the almost complete absorption of animal substances. The digestibility of milk in adults is only slightly less than in infants, which would scarcely be supposed from its composition, but there are probably individual differences.

The absorbability of vegetable substances is far less than that of animal substances. As is shown by the Table, much of this depends upon the mode of preparation. In materials which are baked the addition of coarse bran lessens absorption; finely ground, however, it increases absorption. Cellulose in the potato is readily digested, and best the cellulose of young leaf vegetables. In all of these carbohydrates the absorbability is largely dependent upon the manner of preparation.

As is also evident from the Table, bread made of rye, ground from the whole grain and baked with yeast, and pumpernickel show the lowest degree of absorbability. Among vegetables, the same is true of beans, Savoy cabbage, yellow turnips. It is to be regretted that the degree of absorbability of other vegetables and other varieties of cabbage is not given in Rubner's Tables. The latter, however, may generally be reckoned among the carbohydrates that are only slightly absorbed.

Among substances which exert a mechanical action belong all products with skins, membranes which are usually eaten raw, but sometimes also cooked, and which are very imperfectly digested. Among these are all the different fruits, in so far as they are unpeeled, among the legumes peas and beans, and among other vegetables asparagus in particular. Mushrooms are not at all digested; probably to the same category belongs the radish. Complete absence of digestion also characterizes mustard seed and linseed. These, therefore, are occasionally employed with success as coarse, mechanical, euperistaltic remedies. However, the possibility of the entrance of mustard seed or linseed into the appendix may be regarded as a source of danger.

We note, therefore, from this description that, in a broad sense, the diet contains all the potentials which are necessary to affect a sluggish intestine, and that these are occasionally combined with one another in a most remarkable manner. For example, in fruit we find sugar, acid, cellulose and pectin. The methods of serving these also produce combinations which assist intestinal activity; for example, sour milk with sugar and rye bread, cold cereals, sugar, currants and hyperfermented beer.

We are thus enabled to find a number of laxative remedies from which we must select the best and most effective, and combine them.

It must be primarily emphasized that in cases of advanced habitual constipation that are at all stubborn, any single remedy to stimulate intestinal activity will usually be without effect. If, however, we combine several of these, for example, by administering sugar, acids, table salt, oil, material rich in residue, in proportions adjusted to the mode of life and the habits of the patient, *the total effect equals that of a powerful purgative.*

It is obvious that in the choice of these natural purgatives we must individualize very strictly. In addition to certain idiosyncrasies or bad habits which must be reckoned with in practice, the state of the gastro-intestinal canal comes into question. Acid and fatty substances are frequently not well borne; they may produce pyrosis and a sense of gastric oppression. In other cases coarse bread and pumpernickel generate gas, and other products must occasionally substitute for the varieties of sugar and fruit. Certain disturbances in metabolism, for example, gout, obesity and diabetes, may necessitate a reduction in amount.

This apparent or actual intolerance should by no means force us to abandon the treatment, for the dietetic laxatives which remain may be given in increased quantity. On the other hand, inversely, the diet must be so regulated that secondary disturbances, for instance, anemia, chlorosis, under-nutrition, and enteroptosis may be favorably influenced by it. In such cases the proteids, sugar, and fat should be particularly prominent in the diet.

A significant point in the treatment of habitual constipation is the strict avoidance of astringent substances which inhibit peristalsis. Self-evident as this appears to be, in my experience such errors are frequently committed. Thus, for example, a patient with constipation is placed upon a very suitable diet, but, at the same time, is permitted to take cocoa, red wine, rice or fine wheat bread, or, what is equivalent, these foods are not strictly prohibited. Attention need scarcely be called to the fact that a diet for constipation is thus made absolutely of no effect.

Concerning this somewhat complicated system the question may be and has been asked: Why institute such a tedious treatment when we may more quickly attain the same results with other curative remedies?

Were this actually the case, the dietetic treatment of habitual consti-

pation would, in truth, be an unnecessary hardship. Later on we shall discuss other valuable remedies which increase intestinal activity. But, in considering the results of dietetic treatment, and out of a rich experience, I deduce the following:

The effect of the constipation diet is, in the overwhelming majority of cases, very rapid, and appears, as a rule, even within the first few days. In most cases this result is permanent, and the patient gradually resumes his normal diet. We then consider this an *actual cure.* In the minority, however, after weeks or months the effect apparently ceases; the patients respond imperfectly, or not at all, to the diet. We see, therefore, the same necessity of adapting the stimulation to the case as in a treatment by purgatives. Occasionally, however, this diminution of stimulation is not due to the cumulative effect of the diet, but to the fact that in the course of time the patients fail to adhere to it. Occasionally the treatment is interrupted by prolonged traveling, by improper food in hotels, by psychical emotion or other unfavorable circumstances, and it then becomes exceedingly difficult to stimulate afresh the impaired intestinal function. In conclusion, there are patients in whom a regulated diet acts only so long as they adhere rigidly to all of its details; even slight modifications produce disturbances. In these cases a cure such as I mean is out of the question.

These exceptions, however, cannot alter the fact that the effect is permanent in a large number of cases. *To say nothing of purgatives, such a permanent cure never takes place otherwise nor by any other remedy.*

For this reason dietetic treatment of the mild as well as of the severe forms of habitual constipation must form the normal process. When this is not effective, other remedies are to supplement it, but by no means to substitute for it. Following this is a *diet list* for an ordinary case of habitual constipation, the condition of the stomach being considered normal:

In the morning upon an empty stomach: A glass of salt water or sugar water.

8 o'clock: Coffee with milk, one tablespoonful of milk sugar (or manna or levulose), 50 grams of Graham bread, or D.K. bread, or Simon's bread, 10 grams of butter, 20 grams of honey or sweet marmalade.

11 o'clock: 250 grams of sour milk, or buttermilk, or kefyr 2 days old; 50 grams of Graham bread, or D.K. bread, or Simon's bread; 10 grams of butter (1 egg [1]).

2 o'clock: 50 grams of potatoes (mashed potato or with the skin), 200 grams of vegetables,[2] 150 grams of meat or fish, 50 grams of salad

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[1] With a good constitution this may be omitted.

[2] With a weak stomach as purée; with a good stomach, in the natural form.

(with lemon and oil); about 100 grams of stewed fruit (sweet or sour), 150 to 200 grams of cider, Moselle wine, or lemonade; raw fruit.

4 o'clock: Coffee with milk or whipped cream, 50 grams of marmalade, 25 grams of Graham bread or Graham biscuit.

8 o'clock: 100 to 150 grams of meat or fish (cold or warm); salad (see above); 50 grams of stewed fruit; soft cheese; 50 grams of Graham or rye bread, or pumpernickel with butter; 100 to 150 grams of cider or Moselle wine.

Before going to bed: Raw fruit of various kinds (according to season) and in desirable quantity.

We add here, as an example of a variation in the diet, the following which may be given in a mild case of *diabetes*:

Upon an empty stomach: A glass of salt water.

8 o'clock: Coffee (black); 30 grams of manna, 20 grams of Graham bread, 30 grams of butter.

11 o'clock: 250 grams of sour milk (buttermilk, kefyr); eggs (1 to 3), also scrambled eggs with considerable butter or bacon.

2 o'clock: About 200 grams of vegetables (spinach, string beans, cauliflower, Brussels sprouts, asparagus, artichokes, sweet potatoes); about 50 grams of salad (with lemon and oil), about 100 grams of diabetic stewed fruit; 200 grams of cider or Moselle wine; radishes; butter and cheese according to desire, 25 grams of Graham bread, and two apples.

4 o'clock: Coffee without milk, 50 grams of manna, 50 grams of *bitter* marmalade.

8 o'clock: 100 to 200 grams of meat or fish (even fat varieties, warm or cold); salad (as above) or mayonnaise; diabetic stewed fruit; 50 grams of pumpernickel; 30 to 50 grams of butter; cheese (as desired); Moselle wine; radishes.

Before going to bed: Two apples.

In this diet the amount of carbohydrates is not very considerable, but it may be even further reduced. Manna, which acts as a stimulant even in the severest forms of diabetes, undergoes complete combustion. Attention is called to the large amount of fat in the food which serves the purpose of stimulating intestinal activity.

If there be a predisposition to obesity, the fat should be omitted from the diet, and this, as a rule, will produce a sufficient reduction.

In the given case other curative methods may be added to these dietetic rules, and this will ensure a gratifying result. Although there can certainly be no objection to this, yet, from a critical standpoint, it is advisable first to test the effect of one method, and to employ a second only when the first has failed.

With this constipation diet marked flatulency is frequently observed, even when its effect is otherwise very good. By a suitable change in the details this may be avoided without decidedly lessening the effect. Occa-

sionally, however, as I have observed, a certain self-adaptation of the intestine is noted. The fermentation, previously marked, gradually ceases, and occasionally this cessation appears to be sudden.

As further aids, we most frequently resort to the following: *Abdominal massage, gymnastic exercises,* and *electricity.*[1]

In regard to *the first* of these we must discriminate accurately, as in the results of the diet, between temporary success and permanent cure. That the former is often attained, even with surprising rapidity, there cannot be the slightest doubt. But, in my experience, a permanent cure, even in cases not too far advanced, i. e., those which promptly respond to dietetic treatment, does not so frequently occur. At all events, the cases of cure of severe habitual constipation exclusively by massage are certainly rare. It might be urged in opposition to this that massage was unsuitable in these cases, or, perhaps, that it was not long enough continued. This is, however, not the case. I have seen striking failures after very thorough and long-continued massage.

The results of *electric* (*usually faradic*) treatment are, in my experience, more favorable. For this purpose we employ only faradization of the abdominal walls by means of broad, flat electrodes, or, even better, by means of massage rolls, or, what is perhaps best, intrarectal faradization by means of soft rectal sounds composed of Nelaton rubber.[2] The treatment, at first, should be for about ten minutes daily, and with increasing improvement may be only every second or third day. In the course of time the patients frequently learn to apply the current themselves, or with the aid of some one at home; a factor which is not to be undervalued in comparison with massage.

Whether severe cases of habitual constipation can be cured either by faradization of the abdominal walls or by intrarectal application appears to me to be doubtful. But I can positively assert that the combination of faradization with the diet elaborated above will generally bring about a permanent recovery. Unquestionably relapses will occur and—although rarely—failures. In my experience a point of practical importance is that, if no results are obtained by rectal faradization within the first two weeks, they do not follow even with continued treatment.

As massage and electrotherapy have found numerous adherents, so also have *gymnastic exercises*, indoor gymnastic exercises (Schreber's curative gymnastics) as well as mechanical gymnastics (Swedish curative gymnastics). In how far these methods are permanently effective without the aid of others I cannot state, as I have no personal experience. That they assist in a greater or less degree cannot be doubted.

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[1] It is not our object to enter here upon the technic of these methods, and we limit ourselves to a critical analysis of their curative value.

[2] *Boas,* "Diagnostik und Therapie der Darmkrankheiten." Leipzig, 1901, 2. Aufl., page 178.

Among the methods of treatment most frequently employed, and also misemployed, is *hydrotherapy* in its various modes of application. In the atonic forms of habitual constipation, cold Sitz baths, or half baths (22° to 20° R.) with high affusions over the abdomen, as well as rain baths with a movable stream upon the abdomen, are of use. In the spastic form protracted full baths of warm water (30° to 32° R.) or warm Sitz baths are advised. The warm douche, the Scotch douche, warm compresses to the abdomen, application of hot tubes, steam compresses, and thermophores are all beneficial. In obstinate atonic constipation I have occasionally seen remarkably good results from the application of an ice-bag to the abdomen.

In one case, which was extraordinarily tenacious, there was a profuse evacuation, contrary to all expectation, when the patient (who was suffering from purulent cholecystitis) had an ice-bag applied over the hepatic region. After the cessation of the attack constipation reappeared. On the repetition of the attack of cholecystitis, an ice-bag was again applied, and normal bowel movements again took place.

Such hydropathic treatment is generally indicated in cases of intestinal neurasthenia; but, on careful observation, we must admit that failures under this form of treatment are by no means so rare as some enthusiastic hydrotherapeutists report.

Up to a certain degree, some varieties of sport are included among the physical methods: Bicycling, rowing, tennis, golf and others. They may also assist the curative action of other remedies.

We now come to the important, and most popular, form of treatment: *treatment by purgatives.*

The value of purgatives—of this there is no doubt—is, according to the nature of the case, chiefly symptomatic. Nevertheless, we note in some cases that mild or strong purgatives are taken for years, and even decades, without injury to the patient and—what is more important—they do not lose their effect. As a rule, however, such a permanent effect is not often seen.

Usually the condition is such that patients rapidly exhaust the entire range of purgatives, from the mildest to the most drastic; and it not rarely happens that even these are ineffectual unless taken in dangerously large doses, when, accompanied by severe pain, they produce the desired result.

These experiences which are constantly repeated in tiresome monotony give to the true purgatives a merely secondary position in the treatment of habitual constipation. I employ them only under two circumstances: In cases of constipation so obstinate that dietetic or physical remedies have no, or but very slight, result. But in such cases I also utilize the powerful effect of the constipation diet, and supplement it by the administration of a mild laxative in the smallest doses, or by suitable injections. I use

purgatives extensively in the *constipation of the aged*, i. e., in persons beyond sixty years of age. At this age we may expect to use moderately strong purgatives for years without experiencing difficulty. In the last, or prior to the last, decade of life I also prefer purgatives to injections as being more simple. It is presupposed in this that the patients have not as yet exhausted the whole armamentarium of purgatives.

In regard to the choice of purgatives in the individual case, mild purgatives have for a long time been distinctly separated from true drastic agents. The latter are never employed in the treatment of habitual constipation unless complications occur in the course of the disease. A great number of mild remedies are at our disposal, so great that the physician, no matter how numerous his patients, will scarcely ever be in a position to utilize all the drugs.

In my experience the following remedies are quite sufficient: Rhubarb in its various forms, magnesia, effervescent magnesium citrate, Carlsbad salt, flowers of sulphur, compound licorice powder, extract of easeara sagrada (in the form of the fluid extract or in pill), the preparations of tamarind and, finally, among the newer remedies, purgen (0.5 three to four times daily). Preparations of *aloes, podophyllin, colocynth, bitter waters and bitter salts, castor oil, and many others should be relegated to oblivion in the treatment of habitual constipation: They do more harm than most physicians believe.*

The different laxative teas commonly used by the laity (Gastein tea, Hamburg tea, Hartz Mountain tea) and similar compositions are merely arbitrary and wholly superfluous combinations of various active laxatives, the effect of which must, naturally, vary according to the dose and preparation.

More suitable and simple laxative teas for domestic use are, for example, alder or senna tea (and also cassia); the herbs are placed in cold water and then allowed to boil. I occasionally employ the latter in this form, and it acts very profusely and without causing griping.

In employing either of these remedies it is well, as Einhorn advises, gradually to decrease the dose; this may be done in many, but not all cases. In any event, it should be our object not to use laxatives unnecessarily, and it is wise to continue to use *the same remedy*, even though in increased doses, as long as possible.

Combined with a laxative therapy is the employment of *mineral waters*. Among these sodium chlorid and Glauber salt, occasionally also Epsom salt, play the main rôle. Possibly the action of these spring waters is more or less increased by the amount of carbonic acid which they contain. As representatives of the first group we have the so-called *sodium chlorid waters* (Kissingen, Homburg, Wiesbaden, Mergentheim, Soden). *Glauber salt* is represented by the hot springs of Carlsbad, the cold springs of Marienbad, Tarasp, Elster, and Franzensbad. *Epsom salt springs* may be

employed in stubborn cases, such as those of Kissingen, Friedrichshall, Hunyádi-Janos, Saidschütz, Montmirail, Apenta, etc.

It is evident that mineral water cures are nothing more than purgative cures, and that from these alone permanent relief (in our sense) is not to be expected. Therefore, cases of habitual constipation should not be sent to such spas with the idea that the treatment there forms the main indication. In such cases, the patients return from the fatiguing and expensive journey much disappointed; a condition which I see every season. On the other hand, mineral water cures, like laxatives, may heighten the effect of proper dietetic treatment or may lessen and even remove some of the complications of habitual constipation, for example, cholelithiasis, hemorrhoids, and the symptoms of so-called autointoxication.

Unfortunately, in the realization of this hope the diagrammatic effect of the spa treatment with its monotonous, bland diet is rather inhibitive than effective. In this respect, the spa physician should try to modify the traditional and uniform dietary in force at the different resorts.

Injections form a valuable auxiliary in the treatment of habitual constipation. It is well to differentiate between occasional enemata and *retained* enemata; both have their distinct indications and proper places. The first serve to dislodge by a single, quick stimulation masses of feces impacted in the lower portion of the bowel. For this purpose we may combine chemical or thermic stimulants with the fluid used for injection.

Since, however, in these cases, as the name indicates, the action is to be rapid, medicaments are of little importance. We may rather look for an effect from thermic agents. For this the rule is to employ occasional injections, preferably without additions, about one-half to one liter of cool or cold water. The smaller the quantity used the better the result, as, otherwise, an immoderate distention of the already paretic musculature may easily be produced.

Much more suitable, and probably more efficacious, are *retained* enemata. They are indicated in atony of the entire colon, and, inversely, chemical additions are very suitable, particularly such as have a tendency gradually to soften the fecal masses, while the low temperature may readily cause an abnormally rapid peristalsis, and, consequently, only partial evacuation.

Among valuable retained enemata we must first mention *those of oil* which have been much employed by Fleiner's advice. The action of these oil enemata is not, as might be supposed, coprolytic but antispasmodic. They quiet the abnormally excited muscular action of the intestines, and thus produce normal peristalsis. The chief indication for oil enemata is, therefore, in spastic constipation, although satisfactory results are now and then attained also in the atonic form.

The oil enema should consist of not more than 200 to 300 grams, and this should remain in the large intestine about ten hours. Larger quan-

tities are superfluous, and are expelled more rapidly than is desired. Inversely, we may also obtain results with smaller quantities, and it is therefore advisable gradually to decrease the quantity of oil. Occasionally, from the beginning, smaller quantities of oil (50 to 100 grams) will suffice.

Fleiner attaches great value to a neutral oil, and for this reason advises the purest olive oil. In my extensive experience other less refined oils are just as useful, for example, poppy-seed oil, palm oil, linseed oil, rape oil, and others. Sesame oil is less costly than olive oil, and just as free from fatty acid, while peanut oil, lately introduced into commerce, is good and cheap.

One disadvantage of the treatment by oil enemata is the soiling of the body and bed linen, which cannot be prevented even by the greatest care. An attempt has therefore been made to substitute other active agents for oil enemata. Among these I have advised emulsions of soda, castor oil and cod-liver oil. Soap water may also be emulsified with oil and glycerin, and various additions may be made to these emulsions (castor oil, gum arabic, honey, etc.).

In my opinion it is best to give the retained enema late at night when absolutely at rest, i. e., while the patient is in bed. The injection should be given by means of a simple irrigator and a soft rectal tube. When experience has shown that the oil is not retained, the irrigation tube may be placed in a glass or porcelain vessel so that the escaping oil may flow out without soiling the bed-linen. I have rarely made use of this method.

Instead of an irrigator, large glass syringes, or even more complicated apparatus may be used, but in none of these instruments do I see any particular advantage.

Although the treatment of intestinal peristalsis by oil and emulsion enemata gives excellent results, yet this *by no means effects a cure of habitual constipation.* The method is only palliative. If the treatment is suspended after a short time, intestinal activity also ceases, but unquestionably, as a palliative method, the treatment by oil, although not convenient, is certainly the most effective method. It has the additional advantage that it does not lose its effect by long-continued use, and in this respect is superior to internal laxatives.

Instead of large injections, so-called minimal enemata may be used. Formerly Oidtmann's purgative, which consisted chiefly of glycerin, was considered very useful. In fact, glycerin in small doses (2 to 5 grams) will liquefy fecal masses in the lower portion of the intestine. In 1887, instead of glycerin enemata, I employed glycerin suppositories, i. e., hollow cones made of cacao butter and filled with glycerin, which since then have been much used, and are, unfortunately, often found in commerce in improper form.

Glycerin and glycerin suppositories may have an irritating effect; in

hemorrhoids they sometimes increase the tendency to bleeding, and, under these circumstances, are contraindicated. When there is fecal accumulation in the rectum, however, they are valuable.

Hiller [1] and, lately, Kohlstock [2] have advised for rectal injection the addition of active principles of other laxatives. According to Kohlstock, aloin and cathartic acid, and, in particularly stubborn cases, colocynthin and citrullin should be employed. These minimal enemata are said by Kohlstock to have a prompt and painless effect. But the high price of the remedies unfortunately prohibits their general employment.

The treatment of constipation by means of *subcutaneous injections* has found little favor, but some of the above named remedies and some of the neutral salts (magnesium sulphate) have been employed with more or less success. The same is true of endermatic treatment of constipation by means of croton oil (6 to 10 drops in 15 to 20 grams of olive oil) as advised by Scarbinato.

Furthermore, I must call attention to a mode of treatment of habitual constipation which is apparently paradoxical, nevertheless, in the severest forms, it has produced some surprising results. This is the *rest cure.* I employ this in cases accompanied by general nervous weakness, in which the patient is under-nourished, and in which habitual constipation is the main symptom. In cases which for years had resisted the strongest purgatives, I have seen results in a very short time, usually after even a few days.

The diet is that formerly described. The rest treatment is preferably carried out in a sanatorium, and should be continued for from four to six weeks. Under this treatment the patients usually increase decidedly in weight, which favorably influences the general neurasthenia. By this method, or at least after a very few days, the patient must do without laxatives or enemata, if possible.

Another method of treatment in the neurogenous form of constipation consists in *hypnosis,* which, so far as I know, was first successfully employed by Forel. Lately H. Delius [3] has called attention to the good effects of hypnotism in functional constipation.

From this it is evident that the number of remedies and methods of treatment for habitual constipation is very great, and the list of laxative drugs increases from year to year. In addition, some or even most of these curative methods are used in combination, which makes it difficult to estimate their value.

In my experience the most effectual remedy we possess is a proper diet rigidly adhered to in the manner previously detailed. Occasionally it may be without result, but, in such cases, other curative measures are also

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[1] *Hiller, Zeitschr. f. klin. Medicin,* 1882, IV, p. 481.

[2] *Kohlstock, Charité-Annalen,* 1893, XVII.

[3] *H. Delius, Berliner klin. Wochenschr.,* 1903, Nr. 38.

fruitless. In addition to the diet for constipation, intrarectal faradization of the intestine alone can be considered a permanently useful remedy, and this is, at the same time, one that can be readily used by the patient. Treatment by enemata and suppositories, as a palliative method, and correctly used, is harmless in uncomplicated habitual constipation, and, if the other curative measures mentioned fail, this should first be tried. But the other remedies, whether of chemical or physical nature, can be dispensed with to-day to a much greater extent than is generally supposed. *Treatment by purgatives, now so popular, is, except for a few previously mentioned indications, the survival of a medieval, humoral pathology which we should forever discard.*

TREATMENT OF HEMORRHOIDS

The treatment of hemorrhoids, as is well known, forms one of the many boundary lines between internal medicine and surgery. Therefore, from my internal standpoint, I limit myself chiefly to the conservative treatment of hemorrhoids, and, in so far as operative treatment comes into question, I shall describe the principal indications.

As we have previously seen, hemorrhoids are largely due to local inhibitive causes which prevent the flow of blood into the great reservoirs of the liver (the portal vein and vena cava), and therefore, broadly viewed, the primary object of treatment is the removal of these factors which inhibit the circulation of the blood.

In many cases the carrying out of an etiologic treatment is simply impossible; for example, in hemorrhoids due to pregnancy, in inoperable tumors of the bladder, of the colon, and of the rectum, in marked enlargements of the prostate gland.

Nevertheless, these conditions are decidedly subordinate compared with the most frequent form of hemorrhoids which chiefly owe their development to fecal masses impacted in the lower part of the intestine. Moreover, hemorrhoids are not infrequently accompanying symptoms of catarrh of the colon, even when this latter affection leads to diarrhea.

In delineating the treatment we shall consider first the common form of hemorrhoids, that due to habitual constipation, and here the relations are easily traced. Our first object must be the removal of the cause of the hemorrhoids, i. e., the constipation, and especially the accumulation of feces in the lowest portion of the intestine. There are many remedies and methods for this purpose, but all are not practicable, some are even dangerous. In general, we may state that all treatment by laxatives, not merely by drastics, is inadvisable.

Laxatives, which are at first given in very small doses, soon lose their effect; or, if given in large doses, their action is too powerful and produces unpleasant secondary symptoms. The patient is then in a constant

struggle between incomplete or immoderate evacuations, both unfavorable conditions for hemorrhoids.

The same may be said of the treatment of hemorrhoids by enemata. Frequently the varices become inflamed by mechanical or chemical irritation, and this increases the sufferings of the patient. At most, mild enemata of oil by means of a soft rectal tube which should be cleansed each time before use may prevent these disagreeable consequences.

In hemorrhoids combined with constipation the rational method consists in producing sufficient and properly formed, i. e., pappy, evacuations by a continuous dietetic treatment, and to prevent fecal impaction in the region of the ampulla recti. As this is made possible in the overwhelming majority of cases, even the very chronic ones, by the previously described diet for constipation, there is, as a rule, no indication for treatment by laxatives. Exceptionally this must be considered, either when rigid adherence to these dietetic rules is impossible or when, in fact, even with careful employment, it produces no result. But in these cases, as already stated, small doses of the mildest laxatives will be sufficient—I allude to the old and reliable compound licorice powder, flowers of sulphur, rhubarb, cascara sagrada, and the like.

Besides the special diet, general hygienic laws, so far as practicable considering the occupation of the patient, must be observed: As much exercise as possible, gymnastics, swimming, rowing, billiards and bowling, lawn tennis and football. On the other hand, it is frequently maintained that horseback riding and bicycling increase the distress from hemorrhoids.

The value of these exercises must not be undervalued, but neither must it be exaggerated; in my experience the latter is most often the case.

According to Oeder,[1] a valuable aid in the treatment of hemorrhoids consists in elevating the buttocks; this may best be accomplished by using a wedge-shaped pillow or by the elevation of the foot of the bed, thus causing a better circulation of the blood from the nodule. This is said to be more beneficial than any form of bodily exercise.

Mineral water cures take a prominent place to-day in the treatment of hemorrhoids and are carried out either at home or at the springs. Here, too, the springs which contain sodium chlorid or Glauber salt are the best. As to the results of this treatment we may refer to what has been previously said, in which perhaps the effect of these cures may be estimated somewhat more highly than in the case of uncomplicated constipation, since here simultaneously the favorable influence of the baths is added. But just as habitual constipation is never cured by mineral water treatment or even relieved for any length of time, neither is there any permanent effect upon the hemorrhoids. It is therefore the duty of the family

[1] *Oeder, Zeitschr. f. diätet. u. physikal. Therapie,* 1900–1901, IV, Heft 8.

physician, if such a treatment is under consideration, expressly to warn the patient not to entertain exaggerated hopes.

In the same category as mineral spring treatment is the *grape cure*, which also tends to regulate the intestinal function, and in this manner has an immediately favorable effect upon the hemorrhoids. But, like the treatment with mineral waters containing sodium chlorid and Glauber salt, there is no permanent result.

Another part of the treatment of hemorrhoids consists in the observation and the proper treatment of the nodules themselves.

Observation should begin in the earliest stage of hemorrhoids. Cold applications to the hemorrhoids, solutions of tannin, iodin, potassium iodid or alum are advisable, preferably after a fecal evacuation for the nodules are then most prominent. By this treatment the vascular wall becomes somewhat leathery, and the tendency to hemorrhage and ulceration is lessened. With so-called internal hemorrhoids, the same effect may be produced by injections into the rectum of the previously mentioned solutions.

The etiologic treatment of hemorrhoids is valuable, and chiefly indicated in those cases of hemorrhoids not yet far advanced. It fails of success, however, where the affection has become severe, or where there are complications.

Even in such cases symptomatic treatment is often useful, and may produce permanent, sometimes only temporary, amelioration.

Except for some rare sequels, the *complications* to be chiefly considered are the following: Ulceration, hemorrhage, incarceration, and prolapse.

Mild cases of ulceration may be cured by suitable astringent suppositories consisting of tannin or aloin, or, quite properly, anusol suppositories (bismuth with iodin, resorcin, and sulphurous acid) or by injections of solutions of silver or protargol.

According to Kossobudskj, suppositories consisting of chrysarobin, iodoform and belladonna are much employed in about the following formula:

℞ Chrysarobin 0.08
Iodoform 0.02
Extr. Belladonnæ 0.01
Butyr. Cacao 2.0
M. D. S.: Suppository introduced two or three times daily.

In external ulceration an ointment of the following composition is advised, and is to be used in the same way:

℞ Chrysarobin 0.8
Iodoform 0.3
Extr. Belladonnæ 0.6
Vaselin 15.0
M. D. S.: Applied several times daily.

In severe cases it is well not to spend too much time on palliative methods, but to advise the operative removal of the nodules.

Bleeding from hemorrhoids, if only slight or infrequent, requires no special treatment. When it occurs regularly and debilitates the patient, it must be checked.

In such cases we must first attempt to control the hemorrhages by proper regulation of the bowels, i. e., as far as possible to combat the condition by diet. This is by no means always practicable, and therefore to control the hemorrhages, or, at least, to limit them, we must resort to suitable internal or external remedies. For this purpose, styptics have been advised and employed, naturally in the form of anal injections. It is obvious that these remedies (iron chlorid, tannin, alum, lead acetate, ferropyrin, iodoform, etc.) have very little value, inasmuch as they control only a momentary hemorrhage but by no means prevent its repetition. As a matter of fact, I have never seen success from these styptic remedies in cases of obstinate hemorrhage. On the other hand, excellent results are attained by injections of calcium chlorid into the rectum. I order these in a solution of 10 to 100, of which 10 c.c. are injected once or twice daily by means of a small rectal syringe. I am unable to say from my own experience whether the same results may be attained by gelatin injections, but it is quite likely, since the action of gelatin is due to the amount of calcium salt which it contains. At all events, the injection of solutions of calcium chlorid has the advantage of greater simplicity. Even with this method I have occasionally seen failures or imperfect results; but this is exceptional.

Styptics have also been employed internally, and I must mention particularly the extract of hamamelis which is praised by some as almost a specific in the bleeding of hemorrhoids. I have employed the fluid extract of hamamelis in doses of a teaspoonful three times daily, and in some cases unquestionably with good results. To effect a permanent cure, however, it must be continued for many months. Besides the internal use of hamamelis, suppositories of hamamelis (0.25 per dose) may be employed, but I believe that better results are obtained by its internal administration.

In a serious, acute hemorrhage we should not spend much time on uncertain remedies. The most effective is careful tamponing of the rectum with ferropyrin, iron chlorid, or iodoform gauze. Tamponing with gauze dipped in a 30 per cent. solution of hydrogen peroxid is, perhaps, advisable, for H_2O_2, as I have long known, has an excellent styptic action. With this treatment, however, we must be careful to see that the tampon actually reaches the bleeding point, for, otherwise, the hemorrhage will continue in spite of the tampon. At the same time a decided dose of opium should be given internally to arrest intestinal peristalsis. After the hemorrhage has ceased the tampon may be removed, and the intestine

may be emptied by oil enemata or the internal administration of castor oil.

Another and very serious complication is *incarceration* of the nodules. Usually the physician is called in after many improper methods to produce taxis have increased the incarceration to the utmost. It is dangerous then to continue efforts to replace these nodules, particularly if they are swollen, of an intense blue color, the surroundings edematous, and the patient moaning with pain. Here quiet waiting is preferable to any active measures. We must be content with elevating the buttocks in the manner described by Ceder, the application of ice to the hemorrhoids, and the employment of opium and belladonna by suppository. If the physician is called to the patient during the daytime, depletion by the application of leeches to the areas surrounding the varices (not these themselves) may rapidly produce amelioration and the desired reposition. If gangrene has already set in, we make no attempt to replace the hemorrhoids, but treat the gangrenous nodules with antiseptic powders (xeroform, airol, vioform and others).

In conclusion, we must mention one of the most disagreeable, although not very painful, complications, *prolapse* of the hemorrhoids. In milder cases, i. e., those in which prolapse occurs only upon defecation, the patients soon learn to aid themselves and readily return the prolapse to the rectum. In advanced cases, prolapse follows any exertion, or, at least, any that is considerable, and then it becomes desirable and necessary to resort to preventive measures. Simple remedies, and also occasionally very useful, are the so-called *hemorrhoidal pessaries* (first advised by Dr. Lütje). But these often fail in their action, causing pressure and pain, and are more often carried in the pocket of the patient than in the rectum.

In severe cases of hemorrhoidal prolapse, a so-called rectal support has been advised by Esmarch, i. e., a sort of plate which is placed over the anus to prevent the prolapse. I do not know whether or not this apparatus has proven useful. I tried it in a few cases of severe prolapse, and it was unsuccessful.

As is shown by the preceding, internal and conservative therapy has a wide field in the treatment of hemorrhoids, particularly if the affection is not too far advanced, and if there are no serious complications; but it also has its limits, when the only relief is by operation. This method will be employed more rarely, the earlier and more carefully we institute treatment by the methods above described.

MUCOUS COLIC AND MEMBRANOUS INTESTINAL CATARRH

By G. HOPPE-SEYLER, Kiel

The appearance of membranous, tape-like or circular, even net-like masses in the feces has frequently interested the laity and physicians. The occurrence makes the patient anxious, and gives him an idea that these are parasites, tape-worms or the like, or even that portions of the intestine, particles of its mucous membrane covering, are desquamated. If, as frequently happens, there are severe symptoms, attacks of colic or tenesmus, and only very scant, hard masses are voided, among which are profuse amounts of the abnormal particles, it is not necessary that the patient be of a nervous disposition, as most of these patients are, to make him fear that he is suffering from a severe disease. Neither does the physician regard this clinical picture with the easy assurance which he feels, for instance, in the case of an ordinary chronic catarrh of the large intestine which, in consequence of an improper mode of life, has developed into an acute catarrh with frequent relapses. We are not yet certain of the nature of the affection; a positive etiologic and anatomic foundation is lacking, and curative treatment proves to be difficult, tedious and elusive. Scarcely do we think we have mastered the disease when an error in diet, a psychical climax, any irregularity restores it in all its severity. Nevertheless, the medical world has frequently and very actively busied itself with this disease in the last few decades, and the interest which it has awakened finds expression in the great number of designations which have been proposed for it, and which now abound in literature. According to the special view of its nomenclator, the greatest stress is laid either upon the intestinal lesion or upon the nervous disturbances. We find the following terms employed as synonyms: Enteritis s. colitis membranacea, pseudo-membranacea, pellicularis, tubularis, mucomembranosa, crouposa, fibrinosa, etc., pseudoenteritis, catarrhus desquamativus, diarrhœa membranacea, tubularis, etc., finally, colica mucosa, mucous colic, as Nothnagel[1] designates at least some of the cases. Some of these designations apply only to a few of the cases; the designation "crouposa" or "fibrinosa" does not coincide with what we know to-day of the composition of the characteristic membranous masses. As they are principally composed of mucus,

[1] *Nothnagel*, "Beiträge zur Physiologie und Pathologie des Darms," Berlin, 1884; "Erkrankungen des Darms und Peritoneums," Wien, 1895, p. 144.

the designation "mucosa" is more fitting. Lately, an endeavor has been made to discriminate, and to designate by the terms *enteritis* or *colitis membranacea*, etc., only those cases which present definite symptoms of intestinal catarrh, and particularly of chronic intestinal catarrh. The formation of compact membranous mucus masses is characteristic of this form in contrast to other catarrhs of the large intestine. By *colica mucosa* or *mucous colic*, however, we mean cases which are characterized by the passage of firm membranous masses of mucus in connection with severe attacks of colic. In these we do not assume a catarrhal condition of the intestinal mucous membrane, but believe the condition to be due to a so-called neurosis of secretion. This is favored by the fact that the affection is usually observed in persons who present other distinct symptoms of nervous disturbance.

In the conception of mucous colic as a purely nervous, intestinal disease, we find that it is clinically difficult to exclude a catarrhal condition of the intestine, and that all possible transitional stages are known, ranging from an apparently chronic catarrh of the large intestine with the occasional passage of membranous mucus masses to a well-developed mucous colic without distinctly prominent processes of inflammation of the mucous membrane. In describing the clinical and the anatomical picture we shall have to consider this condition somewhat more in detail.

ETIOLOGY AND PATHOGENESIS

Inquiry into the *cause and mode of development* of the disease primarily reveals the conspicuous fact that the condition usually occurs in "nervous" individuals. The patients invariably show distinct signs of functional nervous disturbance; they are hysterical, neurasthenic, hypochondriac, and often decidedly disturbed mentally; persons with melancholia are especially prone to the attack. These alterations of the nervous system have usually existed prior to the development of the intestinal disease, the certain relation of which to alterations of the nerves cannot be denied. Further, the decided preponderance of females affected is distinctly noticeable. In about 90 per cent. the cases occur in women, the remainder being divided between men and children. Definite relations of the affection in women to diseases of the genital organs have been considered. It is true these very often occur simultaneously with enteritis membranacea; yet a positive connection cannot be determined. If we consider, however, that in consequence of disturbances in the uterus and its adnexa, disorders of the digestive organs frequently occur, particularly constipation, chronic irritative conditions, catarrh of the mucous membrane, mechanical obstruction to the discharge of the feces in consequence of adhesions, displacements, etc., such a condition does not appear remarkable. We must, therefore, assume the predisposition of women with genital affections

to the disease we are describing. It appears, also, that other factors which limit the motility of the intestine, which by torsion or pressure irritate or narrow it, may assist in the development of the disease; thus peritoneal adhesions, bands which constrict the intestine, compressing it and producing traction upon it, must be considered. In cholecystitis, particularly that in consequence of gall-stones, adhesions may readily form which implicate the adjacent intestines. After operations in such persons, we not infrequently note serious disturbances which may be referred to adhesions that have previously arisen. These manifest themselves as disturbances of intestinal activity, particularly by colicky pains, which are frequently believed to be gall-stone colic. Upon daily and close investigation of the discharges, the characteristic mucous pseudo-membranes are either found alone or admixed with hard fecal masses. In connection with an inflammation of the cecum and of the vermiform appendix, perityphlitic adhesions very frequently develop which lead to similar disturbances. Not rarely we find gastroptosis, enteroptosis, nephroptosis, and splenoptosis, etc.; as consequences of lacing, flaccidity of the abdominal walls and emaciation. In some cases the structure of the body has a certain predisposing effect; usually we see this "enteroptosis" develop after moderate pressure by tight clothes and corset has been exerted upon the hypochondrium and epigastrium, or where a ventral hernia has been produced in pregnancy by the over-distention of the abdominal muscles, and this proper support for the organs is absent in an erect position. These processes are often combined with marked loss of fat, loosening of the suspensory ligaments, of the surrounding connective tissue, disappearance of the mesenteric and omental fat, and this loss greatly facilitates displacement of the abdominal organs. This brings the pressure of the displaced kidneys, of the spleen, etc., upon the intestines, particularly upon the large intestine, and, on the other hand, leads to torsion and marked volvulus of the same, which becomes especially operative in the natural bends and flexures of the body. Thus I frequently determined decided displacements and changes in the abdominal organs in women with membranous enteritis, and the difficulties were most pronounced in the region of the ascending and descending colon, particularly in the right and left flexures.

The mode of life appears to have some influence upon the development of the disease. Frequently the regular action of the bowels has been entirely neglected. On account of convenience or social considerations the inclination to stool is disregarded; the intestines become accustomed to the accumulation of feces, and evacuations occur only at intervals of several days. Many women are uninjured by this, and it produces no disturbance of health; in not a few, however, catarrh of the large intestine and a permanent weakness in defecation results. Then, when disagreeable sensations develop, purgatives are taken, without any change in the mode of life, or any attempt to regulate the intestinal activity by exercise, gym-

nasties or massage. Preferably, laxatives and drastics are taken which powerfully irritate the mucous membrane, and stimulate the muscularis to severe, often spasmodic, contractions. Thus an irritative condition of the mucous membrane develops, which does not go beyond the stage of an ordinary catarrh, but which is combined with a tendency on the part of the intestine to irregular, strong contractions, and upon this foundation of abnormal processes, membranous enteritis readily develops.

The disease usually is found in persons of middle life. In advanced age it is rare, and is then the result of a pathologic habit acquired in former years. Not rarely we hear of aged women who had suffered years previously from the passage of membranous masses, and had gradually recovered in later life. In children the affection is rare, yet some undoubted cases are found in literature.

PATHOLOGIC ANATOMY

The *anatomical lesions* in mucous colic and in membranous intestinal catarrh are not yet quite known to us. But few autopsies have been held, and in many of these the question has arisen, in how far the changes found are in actual relation with the affection, and whether they are not to a great extent accidental secondary findings. The disease in itself is not one that endangers life, and therefore is rarely observed at the necropsy; furthermore, we then usually find manifold alterations due to the affection which has caused death. If we omit from consideration the cases not observed during life, the autopsy reports are reduced to merely the cases of O. Rothmann [1] and M. Rothmann.[2] In the patient of the first author, who suffered from typical mucous colic and who succumbed to a perforating ulcer of the duodenum, nothing was found in the intestine. The report states: "Although the entire intestinal tract was investigated, the remaining organs showed nothing abnormal." This does not indicate whether or not a histologic examination of the intestinal canal was made. In M. Rothmann's case the cause of death was a carcinoma at the base of the skull. During life there was obstinate constipation; after intestinal washings numerous strand-like masses of a whitish mucus without fecal admixture were several times discharged. Pain was not present. In this case distinct changes were found in the mucous membrane, particularly in the descending colon. In the transverse colon were some fecal masses. Where these were absent, the mucous membrane was injected and formed in folds, and this was particularly the case in the narrow, contracted descending colon. The narrowed lumen was entirely filled by whitish, partly membranous, partly ribbon-like masses without fecal ad-

1 *Deutsche med. Wochenschr.*, 1887, p. 602.
2 *Deutsche med. Wochenschr.*, 1893, Nr. 41.

mixture. The ascending colon was entirely filled with feces without membranes, but showed reddening of the mucous membrane. The membranes present in the lower portions of the colon and rectum could be detached from the hyperemie mucous membranes without loss of substance. The small intestine was reddened and contained feces. Upon mieroscopic examination the epithelial coat of the mucous membrane was entirely displaced by the mucus masses; they penetrated to the fundus of the glandular ducts, and even gave off lateral branehes to the individual beaker cells. Nowhere could fibrin be found; the stained preparations gave only the signs of mucin. Between the glandular tubules there was cell infiltration and dissemination of the mueosa.

Here we have, therefore, the characteristic features of an intense catarrh. In other cases in which the condition was not observed *intra vitam* the mucous membrane is described as normal, as in the ease first mentioned.

It is evident that in the mucous membrane of the large intestines no gross changes need be present to produce this disease. In very many cases this coincides with clinical observation, for frequently all inflammatory symptoms are absent. When the attack has run its course, no symptoms can be noted. Similar conditions are apparently present in so-called fibrinous bronchitis, as was, for example, mentioned by Litten. But, as in these cases, accurate anatomical data are not at hand.

It is to be hoped that we shall, in the future, secure positive post mortem findings from cases that have suffered from the disease, and thus obtain a clearer idea of the probable alterations of the mucous membrane than is at present the case.

It should be borne in mind that definite conclusions as to the nature of the affection are not to be reaehed in every case in which eompact membranes, or strand-like mucous membranes, have onee bcen passed. Only when the eharacteristic passage of such masses has been observed during life and, as in the case of M. Rothmann, these are still present in the intestine after death, will it be possible to determine the causative anatomical lesion of the affection, and, by the aid of histologie examination, obtain any light as to its origin.

For the present, we must eontent ourselves with obtaining grounds of support for its characterization and pathogenesis principally from the examination of the dejecta and from the elinical picture, and this, as we shall see, is possible to quite a decided extent.

SYMPTOMS AND COURSE

The most important feature of the *clinical picture, and in the course of the disease,* is the passage at irregular intervals of eompaet mucus masses with or without attacks of colic.

The *course* varies greatly. In some cases the disease appears suddenly with a severe attack of colic, and terminates with the discharge of the characteristic masses. These attacks are repeated after days, weeks or months; or it may be that one attack only takes place. In other cases there is at first constipation, sometimes alternating with diarrhea, and this is followed by decided symptoms of tormina, tenesmus, and the passage of membranous masses with or without particles of feces. Simultaneously there is usually anorexia, often a sense of pressure in the head, a sensation of fulness in the abdomen, perhaps even some pain in this region, which, however, is not definitely localized, and does not assume a colicky character. The patients are feeble, are incapable of mental or bodily exertion, and, therefore, readily become irritable and morbid. Evacuation of the bowels, particularly by enemata, brings relief. Laxatives and drastics which they frequently employ, and often to an excess, usually are without effect. On the contrary, these drugs cause decided pain which sometimes resembles colic; tenesmus is increased. The patient's frequent attempts to evacuate the bowels result in the passage of only small quantities of compact or fluid feces, with or without membranes. They become exhausted, and are more miserable than prior to the taking of the purgatives. Rectal injections, provided irritating substances are used, also produce unpleasant sensations and increased tenesmus, and sometimes also tormina. A one per cent. salt solution or Ems water is more grateful to the patient, lessens the tenesmus, and renders easy the expulsion of the mucus masses as well as of the feces that may be present. Frequently the patients take enemata in rapid succession until the abnormal masses and the fecal contents of the lower intestinal areas have been discharged. At first fecal particles are usually passed, then mucus masses with or without feces, finally, often, fecal masses. In many cases the patients anxiously note their fecal discharges. If delayed, or apparently insufficient, enemata are at once resorted to, and if these do not soon produce the desired results, laxatives are employed.

The *characteristic mucus masses* which are voided in this affection assume various shapes. Sometimes they are distinctly membranous and shreddy, at other times they represent solid columns which have, occasionally, a net-like arrangement. The ribbon-shaped, vermiform, or clumpy masses dissolve in water or float, often in numerous, thin, membranous lamellæ. In entwining the mass in which they are rolled together, they tear at many points and are broken up. Upon being shaken in water they separate into numerous flocculent masses. Desquamated intestinal mucous membrane does not have this appearance, nor do parasites, nor connective tissue masses originating from food, or the like.

The mucus masses are usually whitish or grayish-white; sometimes they are stained brownish by the feces, and, according to Nothnagel, in rare cases they are slightly tinged with blood. They are translucent, but not

transparent, as is commonly the case with intestinal mucus. When placed in alcohol the gelatinous masses change to a porcelain white, and become quite tough. They shrink, and do not so readily entwine and dissolve into lamellæ. They may then resemble connective tissue masses originating from food, or fibrinous pseudo-membranes such as are formed in diphtheria.

Upon *microscopic* examination a light, *basic substance* is first noted. This shows manifold fine streaks so arranged that they resemble very thin fibers of connective tissue. On the addition of acetic acid the connective tissue fibrillæ should swell and become indistinct. On the contrary, the striation becomes more distinct and profuse. The striæ form a delicate network, the meshes of which have very sharp contours and are glistening, and include epithelia and round cells. Alcoholic preparations also disclose a coarse, fibrinous basic substance with a deposition of cells, and thus is explained the view of the first investigator of these masses, that they consist of fibrin.

The *epithelia* which are included are usually changed in various ways. Only rarely do they retain their ciliary border; sometimes they are still arranged in rows, therefore detached at one time in large amounts. Some of them still have their normal, cylindrical form; many, however, have enlarged by swelling, and are misshapen, becoming round or more closely resembling pavement epithelium. Others again are elongated and spindle-shaped, so that they resemble smooth muscular fibers, and their terminations are not pointed but rounded off. Fatty degeneration of the protoplasm, the splitting of the cells, as described by Nothnagel, and disintegration up to granular decomposition may be frequently noted; the disintegration of the cells causes them to become friable, and upon pressure on the cover-glass they split up into hard, feebly-glistening, irregular striæ; this process has been referred by Nothnagel to drying, by Kitagawa [1] to hyaline degeneration or coagulation necrosis. Adolf Schmidt,[2] on the contrary, has proved it to be due to imbibition with soaps. Upon addition of caustic potash the cells clear up and the nuclei become more distinct, while an addition of acids does not have this effect. If a particle of the mucus mass is thoroughly admixed with acetic acid or hydrochloric acid, and the preparation is carefully warmed upon an object carrier over a flame, from the cells fine fat globules are apparent which finally collect in large drops under the cover-glass. The cell at the same time clears up, the nucleus becomes distinct; the solubility in ether, and a black staining with hyperosmic acid confirm the fatty nature of the drops which have developed. The cells, in spite of imbibition, are still well retained, for their nuclei

[1] "Beiträge zur Kenntniss der Enteritis membranacea." *Zeitschr. f. klin. Medicin*, 1890, XVIII, p. 9.

[2] "Ueber Schleim im Stuhlgang." *Zeitschr. f. klin. Medicin*, 1897, XXXII, Heft 3 und 4.

stain readily with aniline colors. The epithelia are probably attacked only after desquamation of the fatty infiltration. As the mucous membrane of the large intestine, according to Kobert, secretes fatty acids and calcium salts, and imbibition with calcium soaps, *intra vitam*, would not be impossible, therefore, from what we know of cell alterations of this kind, we can the more readily assume that this process occurs only in the contents of the intestine. The great number of epithelia are indicated by the numbers of those which have a more lamellar character. On the other hand, the solid columnar mucous membrane structures are decidedly deficient in these. The former, to a great extent, are said to be composed of cells, so that intense desquamation of the mucous membrane is to be assumed in their production.

Round cells, in comparison with epithelial, are usually less marked; sometimes they can scarcely be found. A. Schmidt[1] showed them to be more profuse in stained cut sections, and believes they are usually present in larger numbers than is believed, as they frequently disintegrate and in the fresh preparation may therefore be confounded with epithelium. Eosinophiles may also be found among them, with or without the simultaneous appearance of Charcot-Leyden crystals. Plentiful numbers of leukocytes always favor a decided catarrhal or inflammatory process. Masses which consist almost exclusively of pus corpuscles favor ulcerative processes, and in these the mucus intermediary substance is often absent.

Bacteria, which are frequently very numerous in the masses, are of various kinds, and among them are many rods of different shapes, cocci, etc. In many cases hyphomycetes with distinct naked threads are seen. In how far bacteria are the cause of the pathologic process cannot be stated; some authors naturally attribute the disease solely to their presence.

The preparation of dried specimens is not easy, as the smallest particles are difficult to break up; they are not very adherent and constantly slide away; this is probably due to the fat.

Among *crystals* described by Nothnagel are cholesterin plates and those of ammonia-magnesia phosphate. These, as well as the Charcot-Leyden crystals and the fatty acid needles, are present in the mucus masses in very varying amounts and are by no means constant. Remains of food, vegetable as well as animal, are not rarely embedded in the masses.

We arrive at important conclusions by the *chemical examination* of the membranous structures. Naturally this is somewhat difficult, for the masses are but slightly soluble in the mediums ordinarily used for the solution of albumin and mucus products. Cold water will extract some mucin and albuminous products which appear to consist of nucleo-albumin and globulin; these, however, are but small amounts. Weak alkaline solutions extract but little of mucin and albuminates from the masses;

[1] *Loc. cit.*

stronger solutions will dissolve them, particularly upon heating, but they destroy the mucin, and, therefore, no longer convey a clear picture. As A. Schmidt declared, the difficulty of dissolving the masses which, otherwise, are not present in mucus products, depends upon their very intimate infiltration with fat. If the masses are dissolved by remaining for days in a pepsin solution which contains hydrochloric acid, the fluid becomes turbid, particularly near the surface, and fat globules and fat layers are deposited. If these are shaken up with ether the fluid clears, and upon evaporation of the ethereal solution quantities of fat remain. Schmidt has made some fat estimations. In two cases of membranous enteritis he obtained respectively 19.7 per cent. and 9.5 per cent. of fat from the dried substance. More than one-half of the fat consisted of soap, or fatty acids and soap. In a case of true mucous colic, only 2 per cent. of fat was present. Microscopically, the fat cannot be recognized; no needles of fatty acids or soap are to be seen. Unfortunately, the fat is so difficult to extract from the masses that a solution and investigation of the unchanged mucin is impossible; this is usually altered by extraction. The behavior of the masses in a so-called triacid solution after Ehrlich favors the presence of mucin in abundance. They are stained green, as mucus assumes this color. Fibrin or connective tissue, on the contrary, stains red when brought into contact with this mixture. By the red staining of connective tissue masses originating from the food, of desquamated intestinal mucous membrane, and of fibrinous membranes it is possible to differentiate them macroscopically from mucus masses. Schmidt microscopically determined this in sections of stained and embedded mucous membrane masses, and furnished the proof that fibrin was absent. Only connective tissue remains of the food, which are often intimately admixed with mucus, alone show the stains which have been described. Elastic fibers and the remains of vessels are proof, and will protect us from errors. Acetic acid, as already mentioned, produces in the mucus masses a network of strongly refractive threads, which also is in favor of decided amounts of mucin. Only when the membranes contain much cellular material, and, in consequence, become non-transparent, do they clear upon the introduction of acetic acid, in consequence of swelling of the cell protoplasm. In these cases a larger amount of albumin will be found in the masses, and, upon staining with triacid solution, a more pronounced red color will be produced. In spite of the apparently great masses of mucus which some persons with this affection evacuate daily, they lose comparatively little body-substance. A patient of Schmidt's lost only 0.18 grams of nitrogen in the large quantities of mucus which he voided every two or three days. We cannot, therefore, regard these mucus dejecta as reducing the strength of the patient or as producing any marked change in the organism.

Of other symptoms, the *attacks of colic* are the most important; these

frequently dominate the entire clinical picture, while the catarrhal symptoms on the part of the intestine are quite secondary. These are the cases in which, according to Nothnagel, the disease should be looked upon as a nervous affection, and be designated by the term *mucous colic.* The attacks of colic often come on quite suddenly with severe pain in the gastric region (transverse colon) or in the left side of the abdomen (descending colon). The patients give us the impression that they are suffering from a very serious disease; they are often feeble, pale, are very anxious, and we might readily think that peritonitis, intestinal obstruction or the like was beginning. But, after a short or longer time, these symptoms disappear, and usually after the discharge of the characteristic mucus masses, often in enormous amounts. The patients rapidly recuperate and no longer complain of anything serious. Obstinate constipation, however, continues, and sooner or later, often even the next day, a renewed paroxysm occurs followed by the same results. Sometimes the attacks of colic occur only once in a few weeks or months, and the affection may consist of only a single attack. If the disease causes only occasional symptoms, the patient in the interval, apart from nervous disturbances which are chiefly hysterical and a tendency to constipation, are in their usual health, and we are justified in designating this affection, after Nothnagel, as mucous colic or *colica mucosa.*

In other cases there is a very profuse discharge of characteristic membranous and ribbon-like masses, *but the attacks of colic are absent.* There is a tendency to constipation, for the relief of which purgatives are often taken, as the patients, usually women, feel better after a thorough evacuation. The use of purgatives, however, appears to aggravate the disease, and to increase the irritability of the wall of the intestine. The constipation in these cases shows the peculiarity that it apparently runs its course with narrowing of the intestine. This narrowing is usually not organic and due to cicatrices or the like, but probably originates in a firm contraction of the musculature of the intestine; to this marked contraction of the intestine we may also ascribe the sensation which the patients have of tenesmus and fulness in the abdomen. They usually declare that they have the distinct sensation of feces being present in the intestine, which they cannot expel. This irritative condition frequently manifests itself by hunger, which, however, is usually soon satisfied. The intake of nourishment is often scanty, as it reflexly produces more decided irritation of the musculature of the intestine and aggravates the discomfort. Eructations are frequent in consequence of involuntary swallowing of air. Many patients resort to purgatives, others administer one enema after another. After the latter, as a rule, nothing is voided immediately; the water is discharged unchanged. Then hard particles of feces appear which in form usually resemble the feces of sheep. Between these masses, or discharged in separate movements, are the characteristic stools. When a large quan-

tity is voided, this often terminates the attack; the patients have a feeling of satisfaction and relief after such purgings, but they are usually weak and debilitated. In these cases of *enteritis membranacea* which run their course without colicky pains, a more or less decided contraction of the large intestine appears to be present. Sometimes with a very flaccid abdominal wall, particularly upon the left side, the organ may be felt as a firm column. But the contraction is not so spasmodic or so excessive as in those cases which are designated mucous colic, and are often attributed to a neurosis of secretion. There are, however, all possible transitional stages from a dull sense of fulness and pressure with a feeling of severe tenesmus and contraction up to sharp and colicky pains. The same conditions are observed in the common diseases of the large intestine, and also after the administration of laxatives. It must be borne in mind that nervous and hysterical patients offer but very slight resistance to such sensations, that their reaction to the irritation of pain is much more ready and much stronger than in normal persons, and we are almost invariably dealing with neuropathic individuals.

If we compare the sensations in the intestine with those which are produced in severe contractions of the muscles of the extremity, where also painful spasms occur (for example, in the muscles of the calves in consequence of over-exertion, circulatory disturbances, etc.), we note that the same causes often produce only a contraction of the muscles without pain. One patient will describe these spasms as very painful, while another reports them as only slight. Therefore, a strict differentiation of the forms of colica mucosa and enteritis membranacea *cannot* be made, even though we recognize the causal influence of the nervous temperament in the well-developed cases of mucous colic, as was first done by Nothnagel. It does not appear likely that the mucus gives rise to the colic. The view appears to me more reasonable that the mucus is profusely permeated with epithelium, and often coats the scybala in a characteristic, firm, membranous mass, and that the irritation of the mucous membrane is due to a catarrh, to irritative contents, and more or less severe contractions of the muscularis, which compress the mucus produced by the epithelial cells, and form it into columns and bands, as has been accurately described by Marchand.[1] At the same time the mucus becomes deficient in water, more compact, and infiltrated with fat which probably originates from the intestinal contents adherent to the surface of the mucous membrane. These masses of feces discharged show the effects of this compression and deficiency of water; they are friable, and in the haustra of the colon are formed into small or large balls. Although other symptoms of catarrh of the large intestine are lacking, nevertheless, the presence of numerous epithelial cells, sometimes also of round cells in the mucus, is in favor of

[1] *Berliner klin. Wochenschr.*, 1875.

catarrh, and the production of mucus is explained, as well as the irritation of the intestinal wall which leads to colic and to spastic constipation; and these are much more fully and more naturally explained than by the very problematic hypothesis of a mucus neurosis of secretion. That the affection generally occurs in nervous, hysterical women naturally favors the view that abnormalities of innervation here play a part. If, however, there is a more decided irritation of the intestinal nerves, this generally expresses itself in the muscular functions of the intestinal wall. In excitement, in fright, under psychical emotion, as is well known, spastic contraction of the intestine or increased peristalsis readily occurs, and this is particularly marked in chronic catarrh. Here, in excitable persons, spastic contraction of the large intestine and ensuing constipation arise, which are readily relieved by the administration of preparations of belladonna, etc. In the disease under consideration we are dealing usually with easily excited, sensitive persons, in whom an irritative condition of the intestine very readily expresses itself by spasm. If this occur in the intestine, it is followed by a narrow constriction and very decided pressure of the mucous membrane which is now stimulated to a profuse secretion of mucus. On account of a lack of reliable reports of anatomical investigations, and the difficulty involved in the observation of the intestine during the attack, it is as yet impossible to explain with certainty the nature of the affection; but I believe that the above solves the question of the origin of the characteristic mucus formation, and that the other symptoms of the disease may be brought into relation with this without accepting a doubtful hypothesis.

PROGNOSIS

In regard to life the prognosis is generally favorable; but the disease may lead to a lowered state of nutrition and loss of strength, and thus result in chronic invalidism. As a rule, however, the patient attains old age, and later gradually outgrows the affection. The disease may also be cured by proper treatment, combined with good nutrition and the regulation of the mode of life; usually, however, there is a tendency to relapses. In some cases the disease exhausts itself in a few paroxysms, and, in the first attacks, this leads us to hope that they may be the only ones; decided increase in weight, improvement of the general condition, and the disappearance of other nervous disturbances enhance the hope of cure.

DIAGNOSIS

The diagnosis is easy when we find in the dejecta the typical, membranous, ribbon-shaped, net-like or columnar mucus structures. In nervous persons who now and then suffer from severe attacks of colic, from tenesmus and constipation, it is well to examine the feces, and to ask

the patient, or those about him, to observe whether masses of this kind are discharged. Cases of constipation and intestinal catarrh which run their course with colic will then be recognized as intestinal colic or membranous catarrh. It is true we must guard against error in confounding these masses with particles of connective tissue such as originate from meat and other constituents of the food. Frequently, connective tissue masses containing globules of fat which, by the effect of digestive juices, have become saponified and have therefore a whitish color, will be taken for membranous mucus masses. I have often seen them after the eating of raw ham, and they have been shown me by anxious patients or their physicians. They closely resemble the mole-like mass which Rosenheim depicts in his text-book in the chapter devoted to enteritis. By their tenacity when we attempt to separate these masses and entwine them, and the microscopic evidence of particles of transverse muscle between the fibers, and the clumps of whitish, fatty tissue, we may easily differentiate them; moreover, they do not show the reaction from mucus mentioned above, nor do they present coarser connective tissue fibrillæ with remains of vessels, elastic fibers, etc.

Besides tendons and fascia, the lumen of arteries and veins, remains of oranges, milk coagula, masses of fungi having membranes, such as the oïdium, may be confounded with mucus dejecta, but the chemical and microscopical investigation of these objects will enlighten us.

The typical mucus masses are disentangled upon shaking in water and are dissolved into countless small particles, which, upon standing, again separate from the turbid fluid and collect upon the surface. This does not happen with the particles of food, etc., referred to above.

Admixture of blood in the mucus masses favors ulcerative processes; therefore, either dysentery or tuberculous disease of the intestine, deeply invading follicular ulceration or ameba enteritis may be simulated.

TREATMENT

The treatment must be designed to combat the various factors which have been operative in the development of the disease, and which are the individual components of the clinical picture. It must therefore control the irritation of the intestinal mucous membrane, the motor intestinal disturbance, and, particularly, the general changes in the nervous system, the hysterical, neurasthenic and hypochondriacal symptoms. This may be attained by careful regulation of the diet, for, on close investigation, we usually find here much at fault! Often the patients, from a fear that certain food will disagree, limit their diet too much. Mechanical and drug treatment of constipation must also be instituted. All irritation of the intestine should be avoided, and when there is decided evidence of bacterial decomposition of the intestinal contents, the administration of dis-

infectants is indicated. All means to regulate and strengthen the nervous system are, in general, to be employed to the fullest extent.

As the clinical picture varies greatly in the individual patient, at one time this, at another time that, symptom becoming more prominent, special attention must be given to the intestinal catarrh and the accompanying constipation; in other cases, however, in which the catarrhal symptoms are less marked, and a more or less purely nervous affection, therefore Nothnagel's mucous colic, appears to be present, the main stress must be laid upon the regulation of the mode of life so as to avoid deleterious effects which might react upon the central nervous system,—the psychical element. All means must be employed to strengthen the tone of the nervous system.

If examination reveals a well-developed *catarrh of the large intestine*, this must first be treated, primarily by *laxatives*, such as castor oil, rhubarb and the like, thoroughly to cleanse the intestine of long-retained, irritating masses, the generators of fermentation and decomposition. For the latter calomel, a single dose of 0.3–0.5 grams, or, upon continuance of the bactericidal action, the dose repeated for several days, or a dose three times daily of 0.05–0.2 grams will be serviceable. To empty the intestine we employ purgatives, and we assist their action by the use of injections in a manner to be later described; the laxatives render the intestinal contents fluid and propel them, but, frequently, in the lower part of the intestine, this effect is lost, and the fecal masses collect here and are not discharged. The prolonged use of purgatives and also mineral waters, such as the purgative water and springs containing Glauber's salt, is not advisable, as these often have a most unfavorable effect upon the motor irritation and, in general, upon the nervous condition. Therefore, a treatment at Carlsbad, Marienbad, etc., or at the saline springs, such as Homburg and Kissingen, is generally not so beneficial as in a simple chronic catarrh of the large intestine.

Sometimes the use of *astringents*, bismuth subnitrate or bismuth salicylate, and in cases of decided flatulence magnesia, is also of value. These remedies may be combined, or an extract of hyoscyamus or oil of peppermint and the like may be included to allay intestinal irritation.

In the removal of the *motor disturbances* in the intestine, careful massage will be found serviceable in the interval between the attacks, and the application of the faradic or galvanic current, cautious gymnastic exercises for the trunk and leg, moderate physical exercise, such as walking, horseback riding, bicycling, to combat the frequently coexisting atony of the intestine. In such instances laxatives should be avoided as much as possible, *enemata* taking their place, and where there is decided irritability of the intestines, warm injections of Ems water, or a ¾ per cent. saline solution in quantities up to one liter. Previous to this olive oil may be injected, provided the intestine is but slightly sensitive. Enemata of ½ a

liter of oil, according to the methods of Kussmaul and Fleiner, are also valuable. The addition of irritating astringents or disinfectants, such as silver nitrate, tannin, corrosive sublimate and the like, had better be avoided. Drugs which have an anodyne effect, as oil of peppermint, starch, etc., may be added to the injections of water, or the injections may be of chamomile or peppermint tea. These intestinal washings act especially well during the attack; they assist in discharging the hard scybala and masses of mucus, as well as the flatus which inconveniences the patient greatly, and thus serve to quiet the intestine. In the interval between the attacks, preparations of strychnin are best employed, such as the tincture of nux vomica with tincture of rhubarb, which mildly stimulate the intestinal movements; during the attack of colic the belladonna preparations, such as extract of belladonna, in doses of 0.02–0.04, or atropin sulphate, etc., afford relief. Even where the spasmodic contractions of the intestine are painless, these drugs are to be recommended. Morphin and opium should only be employed in cases of necessity. To allay the spasmodic pain, heat may be applied in the form of cataplasms, such as may be combined with the use of the Quincke heat apparatus, or as hot woolen cloths, thermophores, etc.

In cases of a more catarrhal character, therefore, in enteritis membranacea, the *diet* should be non-irritating, and acids, spices, fats, foods rich in cellulose, and those which ferment readily, should be avoided. But this restriction in diet must be only until the marked catarrhal symptoms, such as periodically appearing diarrhea, fermentation, and profuse gas formation, are removed. As a rule, a strengthening and nutritious mixed diet is more stimulating and serviceable in the treatment. A continuous filling of the large intestine with cellulose and other remains of food decidedly stimulates peristalsis without leading to spasm, which readily occurs in an empty intestine on account of the difficulty of propelling the compact mucus masses. For this reason plentiful amounts of bread are usually ordered, not fine wheat bread but the coarser varieties which must be well baked; vegetables rich in cellulose, as well as berries and fruits; meats which contain fat, also bacon and butter. Patients are usually anxious to avoid such a diet, for they believe their intestines to be much too sensitive to retain this food, and, if a few attacks recur, it requires great powers of persuasion to induce them to resume this diet. All strongly spiced food, as well as cabbage and other vegetables which are apt to generate quantities of gas, must be excluded, for this accumulation of gas in the intestine causes great distress to the patient, and readily induces colic; the plentiful ingestion of fat and carbohydrates has the further advantage of increasing the accumulation of fat in the body, which should be particularly borne in mind in the case of nervous, thin individuals who have a downward displacement of the stomach, intestine, kidney, etc.; that is, when enteroptosis is present. The fact that in this condi-

tion the organs are better supported, that the large intestine has a firmer hold upon the mesentery and omentum rich in fat, that torsion of the organs and their nerves is lessened, and movements of the colon are increased conduces to improvement. In cases where the marked effect of lacing exists, proper corsets should be worn and the dress so arranged that the weight of the clothes will rest upon the shoulders rather than upon the abdomen. In cases of pendulous abdomen with marked flaccidity of the abdominal walls after pregnancy, or from a previous abdominal tumor, etc., the wearing of an elastic abdominal bandage is advisable, prolonged standing or sitting is to be avoided, and there should be frequent rest in the recumbent posture.

In controlling the *nervous symptoms*, general rules must be enforced which cannot here be described in detail. This necessitates careful attention to the complaints of the patient, an accurate study of his mode of life, his manner of thought, and the possible unfavorable factors in his occupation, his family relations, etc., which excite and disquiet the psychical sphere, shock the nervous system, reduce the normal resistance to pain and unpleasant sensations, and frequently lead to nervous tension. Sufficient rest at night, occasional interruption of the day's activity by intervals of rest, and especially a certain freedom in diet must be insisted upon. Detailed hygienic instruction by the physician will often relieve these conditions. In cases of necessity, change of climate, residence in a quiet place in the mountains or at the seashore, will be serviceable. Exciting life at summer resorts or at the seaside is to be avoided. These measures may be assisted by carefully chosen local hydriatic procedures, such as cold rubbings, baths, or douches, general massage and faradization, to the abdomen. Finally, as previously mentioned, a generous diet consisting of fat and carbohydrates will be useful in promoting the accumulation of fat in the body.

In general, the treatment of enteritis membranacea and mucous colic is not easy; but a careful study of the intestinal and nervous disturbances present as well as of the circumstances of the patient, and the investigation of all the varying symptoms, may enable us in many cases, with the patient help of the sufferer, to cure the affection, or at least to alleviate it, so that he has intervals of relief from the affection, and it does not so decidedly interfere with his occupation.

INTESTINAL CONSTRICTION AND INTESTINAL OCCLUSION

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I. INTESTINAL CONSTRICTION

The two clinical histories which we shall here analyze and discuss belong to the severest forms of disease with which the physician is brought into contact; chronic constriction of the intestine in the one case, and acute occlusion in the other. I shall detail the pathology of these two conditions as definitely as brevity will permit, and shall first describe stenosis, then intestinal occlusion, proceeding from the simple to the more complex.

The first patient, a woman aged 37, states that she has had five normal labors, and, with the exception of an acute febrile articular rheumatism in her eighteenth year from which she recovered, has always been well. Three months ago she began to suffer from constipation, while previously the bowels were normal. This symptom developed quite insidiously without any preceding change in the mode of life of the patient. She was compelled to take purgatives, which formerly had never been necessary. In addition there was pain of a colicky character, which occurred daily, never lasted more than a few minutes, and frequently subsided with the passage of flatus. Moreover, the patient had observed that, for about five weeks, during the attacks of pain, the intestines and different parts of the abdomen bulged and formed prominences (as she expressed it). In the last three months she has emaciated appreciably.

The totality and grouping of these symptoms are characteristic, and must at once arouse a suspicion of intestinal narrowing. Constipation developing insidiously without recognizable external cause, frequent pain of a colicky nature, and, above all, the bulging coils of intestines, form the characteristic features. The symptomatology of intestinal constriction is simple and comprehensive: The clinical picture is dominated by the fact that the propulsion of the contents of the intestine meets resistance at a certain point due to some anatomical obstruction.

SYMPTOMATOLOGY

Chief among the *symptoms* are *alterations in the fecal movements.* Constipation develops slowly and gradually, since the affection which causes

the stenosis also almost invariably arises insidiously. This kind of constipation is important in diagnosis for the reason that it occurs in persons who have previously had regular evacuations, and no other cause can be assigned for its presence. At the onset, according to the situation of the stenosis, mild therapeutic measures are sufficient for its amelioration—purgatives by the mouth or irrigations per rectum; gradually, however, other symptoms appear while evacuation of the intestines becomes more and more difficult.

The degree of intestinal activity is pre-eminently influenced by the seat of the stenosis; therefore, while the contents are still fluid or semi-solid in the jejunum and ileum their passage is not greatly interfered with, or, at least, only late in the course of the affection; on the other hand, this is marked in the lower portions of the large intestine. It happens occasionally in stenosis of the colon, even in advanced stricture of the sigmoid flexure, that the daily evacuations continue for a long time to be regular.

Occasionally, constipation is interrupted by the appearance of diarrheal discharges, and then enormous masses may be passed. This diarrhea is the consequence of irritation of the walls of the intestine above the stenosis by the retained contents and may persist for a long time. Blood and pus are sometimes admixed with the dejecta, originating, in rare instances, in a secondary decubital ulcerative change in the mucous membrane above the stenosis, more frequently due to definite anatomic forms of stenosis, particularly carcinomatous changes. Special importance in diagnosis is often attributed to changes in the shape of the fecal masses (*feces of stenosis*) : small balls resembling sheep feces, containing one or two grooves; or thin, lead-pencil-like, or flattened ribbon-like, columns of feces. Formations of this nature are not often observed, but, in such cases, they cause confusion for they may occur without any anatomic change in nervous spastic constipation or in cases of so-called starvation intestine.

These changes in the feces, considered individually, are of slight diagnostic value, and this is also true of the other symptom which is sooner or later added, namely, the *colicky* attacks of *pain*. The intensity of these pains varies greatly; sometimes they are extraordinarily severe. In character they are like other paroxysms of intestinal colic. Their seat is generally the region of the stenosis, and from this the pain radiates to other areas of the abdomen; rarely are they more severe anywhere than at the point of their origin. Gurgling, rumbling sounds are often noticed during the paroxysms. Their physiologic foundation is the increased resistance to the propulsion of the intestinal contents, and the decided intestinal contractions thereby produced.

One of the most significant symptoms, often decisive in diagnosis and occasionally pathognomonic, is the *plastic prominence of the tetanically dilated, tense coils of intestines*, with or without visibly increased peristal-

sis. This tetanie, visible, and palpable phenomenon, designated by me as *intestinal stiffening,* finds eonditions for its development only in the presence of intestinal stenosis, with aeeumulation of intestinal contents in a gaseous or fluid form, with energetic eontraction of the intestine as a result of hypertrophy of its walls, and resistanee to the propulsion of the intestinal contents. We distinctly see and feel how a portion of the intestine (perhaps already distended with gas but still soft) is ehanged, usually quite suddenly, into a ridge standing out prominently and rigidly above the surrounding area and aetually to be grasped by the fingers, which remains in this condition for a short time, and then returns to its former elastic state. After a few minutes this is repeated. The patient suffers severe colicky pain which is produced by the tetanie eondition of the intestines. For the diagnostic employment of this phenomenon, it must be shown that tetany does not occur in an empty eoil, or one that ean be emptied, but in a dilated coil, one filled with gas and intestinal contents, which, on account of the obstruction (stenosis or oeelusion), eannot relieve itself.

Besides intestinal stiffening, *increased peristalsis* is often present, and is manifested by a tortuous, more or less aetive forward motion, visible over limited or extensive areas.

The symptom just mentioned (with ehangeable conditions) is eharacteristie of intestinal eonstriction; all other symptoms oeeurring in a concrete case are ineidental consequences, or due to the nature and anatomical composition of the stenosis. The external condition of the abdomen varies as much as the results of pereussion; both are determined by the fulness of the intestines, be it with gaseous or compaet or fluid contents, which vary from day to day or from hour to hour. General diagnostic rules eannot be formulated. One eondition must be briefly described; occasionally the fluid mass which has accumulated above the stenosis becomes so massive that the greatly dilated coils (on aeeount of their weight) drop from their horizontal position to the sides of the abdomen, thereby simulating ascites, espeeially sinee, with this change in position, the full eoils of intestines may also ehange their location. I have observed in stenoses of the sigmoid flexure as well as of the ileum that coils of the small and also of the large intestine may fall into the flanks. A very simple maneuver will decide in sueh cases whether aseites or the fluid contents are present in the lumen of the small intestine. If a few sharp taps are made longitudinally upon this area, dull upon pereussion, and a splashing sound is produeed, it is eertain that fluid is inside the lumen of the intestine.

In the ease whose history has just been detailed the eharacteristie symptoms were present and the diagnosis of enterostenosis eould be made with absolute certainty, but this is not always the ease. The diagnosis is sometimes simply impossible, if no obstruction can be felt, and when

the contents of the intestines are still expelled, either because very fluid, as in the small intestine, or because the obstruction is not yet sufficiently advanced. Between these extremes is a series of cases which seem more or less probable, but which necessitate great care in the diagnosis. A positive decision in a given case depends upon two things: either we must feel the stenosis directly in the rectum—that is, we must palpate a tumor, a peritoneal band—or, upon the surface of the intestines, we must detect a special configuration, the tetanic intestinal rigidity and increased peristalsis during an attack of colic.

What lends to the rigid, chronic stiffening of dilated coils its great diagnostic value, particularly for stenosis? The anatomical change above the narrow area furnishes the explanation of this. The intestine is here dilated, usually only to a limited extent and moderate degree, but sometimes over large areas until an extreme degree is reached. Macroscopically, the distinctly visible muscularis of the intestine is hypertrophic, in cases that are prolonged often decided, increased over great areas, for instance, in stenosis of the sigmoid flexure, up to the ileo-cecal valve. This thickening of the intestinal wall, which in acute stenosis may even be recognized upon the fourth or fifth day, is the product of a true hypertrophy of individual fibers and muscles which owe their origin to increased muscular exertion that is absolutely necessary to propel the intestinal contents through the narrowed area. And as such a marked muscular hypertrophy, capable of developing an enormous rigid stiffening, only arises in extreme stenosis, the diagnostic importance of intestinal rigidity is clear.

ETIOLOGY

We now turn to the question: What etiologic factors are capable of producing such a change in the intestinal lumen that the propulsion of the contents becomes difficult? This condition we designate as constriction of the intestines (stenosis) in contrast to intestinal obstruction (occlusion) in which the lumen is completely closed, and any propulsion of the contents downward, i. e., in a physiologic direction, is absolutely impossible.

The form of, and the causes which may produce, narrowing of the lumen of the intestine, are manifold.

We should digress far beyond the limits of this article if we attempted minutely to investigate these, and must content ourselves with their simple enumeration, giving to each the brief consideration necessary for our concrete purpose.

First, it must be stated that the condition designated as intestinal stenosis is, in a clinical sense, always due to anatomical changes and relations. Certainly, a spastic contraction of the musculature of the intestines will cause a narrowing of its lumen. This, however, is but a temporary

condition, and does not produce the clinical picture characterized by the term "intestinal stenosis." The fecal masses which accumulate in the intestine, especially in habitual constipation, also narrow the intestinal passage; but in this condition the picture of intestinal stenosis with muscular hypertrophy and tetanic stiffening does not develop; should any serious local consequences arise, the symptom-complex of complete intestinal occlusion results.

Chief among the causes of stenosis of the intestine are the following:

1. *Carcinomata of the intestine.* These are almost always primary; only very exceptionally does a carcinoma develop in the intestines by way of metastases, through the blood, or through the lymph-vessels. Naturally we must consider very differently those not infrequent cases in which the neoplasm proliferates from a neighboring organ (the stomach, the uterus, the gall-bladder) to the intestines.

Universal experience teaches us that carcinoma in the small intestine is very rare, is frequent in the colon, and most frequent in the rectum. To illustrate, we will mention only those figures which are taken from the autopsy reports of the Pathological Institute of the Vienna General Hospital. In twenty-four years, among 41,838 autopsies there were 343 intestinal cancers. Of these 7 were in the duodenum, 10 in the ileum (none in the jejunum), 164 in the colon, and 162 in the rectum. Accordingly—and other autopsy statistics agree in this—the number of colon and rectal carcinomata is almost equal. In contrast to this, the statistics in the living show the very extraordinary preponderance of rectal cancers, 80 per cent. of all intestinal cancers being here included. The discrepancy in these statistics may probably be largely explained by the fact that cancer of the rectum is much more susceptible to diagnosis and more readily operable, so that many carcinomata of the rectum are operated upon, and do not come to autopsy in the Hospital.

Clinically, the fact is noteworthy that cancer of the intestine occurs with relative frequency in the first half of life, usually developing before the fortieth year. The anatomical peculiarity of intestinal cancer, that it often distributes itself in a markedly annular form, occasionally encircling the intestinal lumen like a cicatricial ring, makes it possible for the neoplasm to produce decided narrowing which may be so extreme that only a thin pencil or a sound can be introduced. The same stenotic effect, however, may occasionally be produced by all other forms of the neoplasm.

2. *Sarcomata* may also narrow the lumen, but this effect is only rarely observed. On the contrary, in this form of tumor, the intestinal passage is but little influenced; on account of the peculiar distribution of the neoplasm mass into the musculature, the intestinal wall rather becomes paretic.

3. I include here *tuberculomata* of the intestinal walls, which, like car-

cinomata, and in identically the same manner, may bring about stenosis. I have observed several cases of this form of tumor and the literature of the past year also furnishes examples.

4. *Benign intestinal tumors* (myomata, lipomata, angiomata, fibromata, adenomata) only exceptionally form the cause of stenosis; their entire composition and anatomical formation is but little conducive to this and their occurrence is quite rare. At the most they indirectly cause a limitation in the passage by becoming the starting point of an invagination as pediculated polypi. The majority of benign tumors which, by direct limitation of spaee, cause stenosis are situated in the rectum and usually occur in children.

5. *Internal cicatricial strictures* are produced from connective tissue, from cicatrices of ulcers. Of the many varying etiologic forms of ulcer in the intestine, few terminate in a cicatrix which causes a stricture. The most frequent are the tuberculous and stercoraceous ulcerations, more rarely the round ulcer of the duodenum, the dysenteric and catarrhal follicular ulcerations, the syphilitic forms of disease, finally, traumatic lesions and enteric fever ulcerations. This scale of frequency is different from that commonly given, in which the dysenteric cicatricial ulcers occupy the first place, and the tuberculous and typhoid ulcers the last. According to the comprehensive statistics compiled by Woodward, we can hardly doubt the rarity of dysenteric strictures. On the other hand, König has called attention to the fact, opposed to former views, that tuberculous cicatricial strictures have been frequently observed, and this coincides with my own personal experience.

Tuberculous cicatricial strictures occur mostly in the small intestine, particularly in the ileum, as do also the very rare typhoid strictures; those due to ulcer of the duodenum are naturally found in its region; dysenteric and stercoraceous (decubital), in the colon and rectum. The cicatricial stenoses after catarrhal follicular ulcers, which are also exceedingly uncommon, are met with in the small as well as in the large intestine; those due to syphilis preponderate in the large intestine, more accurately, in the rectum. This latter region, more often than any other part of the intestine, may become stenosed by cicatrices which originate from trauma,—operations for prolapse of the rectum, destruction of hemorrhoidal nodules, injury from syringes or introduced foreign bodies, and to these perforating periproctitic abscesses are superadded. Cases of stenosis of the intestine are rarely observed, and up to this time have been exclusively in the small intestine, after severe trauma of the abdomen which has produced a lesion in the wall of the intestine. These anomalies have a purely clinical interest from the fact that when a portion of the intestine is implicated in a strangulation, or after the desquamation of an intussusceptcd portion of the intestine in invagination, a stricture may subsequently develop.

6. *External constriction of the intestine by peritoneal processes*, the anatomical appearance of which has been portrayed by Treves. It is impossible for us here to give a detailed description; only the most important general features will be emphasized. In this etiologic group we invariably deal with chronic peritoneal adhesions, indurations or bands, which have made a portion of the intestine adherent either to another part of the intestine, to any area of the abdominal walls, or to another organ, and, in the latter case, to one normally in the neighborhood of the intestine or abnormally situated there.

In very different ways the lumen of the intestine may be occluded by the action of chronic peritonitis. Peritoneal indurations which are isolated and circumscribed usually encircle the intestine in a ring-shape, or, at a circumscribed point on the lateral surface, the adherent intestine becomes more or less decidedly twisted, when we have a volvulus of the intestine. Or, a general peritonitis leads to concretions, partly of the intestinal coils among each other, and partly of the parietal serosa with the solid abdominal organs. Occasionally, constriction occurs as the result of peculiar extension of the intestinal wall by traction of a diverticulum.

I must refrain from the enumeration of the various anatomical causes and the etiologic modes of development of this chronic adhesive peritonitis, and shall only state that this etiologic form of stenosis may affect the different portions of the intestine quite irregularly.

7. *The intestine may be constricted by a tumor or by any other mass acting similarly by pressure* (gravid uterus, dislocated organs, inflammatory abscesses). In this group those portions of the intestine are most frequently occluded which, on account of their anatomical position, and their tense, mesenteric fixation, are least able to elude pressure; first, the rectum, then the descending colon and the sigmoid flexure, the duodenum, the ileum infimum, the upper portions of the ileum very slightly, the jejunum and the transverse colon.

Besides the factors which have been mentioned, in very rare cases other conditions may bring about a constriction of the lumen of the intestine, such as a chronic invagination, or even sometimes an obstruction from hard fecal masses, gall-stones, or a foreign body. But this is exceptional; for the most part the conditions enumerated are the causative ones.

We now return to our special case with the question: What causes the stenosis in this patient? In which portion of the intestine is it situated? I will recapitulate briefly the important results of our investigation: The patient is afebrile; moderately emaciated; there is pallor of the face; the radial pulse is normal; there is constipation and the previously mentioned abdominal pain. Upon inspection the upper portion of the abdomen shows nothing abnormal. It is situated below the level of the thorax; the lower half, from the navel downwards, is somewhat inflated; there is no meteoric bulging of the flanks. In the lower half of the abdomen,

occasional tetanic intestinal rigidity and progressively energetic peristalsis are noted. Most decided bulging, in extent that of a dinner-plate, is periodically observed in the ileo-cecal region. This area is also the seat of the severest pain. These phenomena appear spontaneously, causing pain which ceases after a few seconds, at most after a few minutes. Occasionally loud gurgling and rumbling are heard. Stress must be laid upon the following: If the abdominal surface is perfectly quiet, it is sometimes possible to produce peristalsis and intestinal tetany by gentle palpation, but, more certainly, by short, energetic taps which invade deeply or by flagellations with a cold, damp towel.

The seat and the relief-like arrangement of these characteristic signs must be accurately studied; as their presence permits the diagnosis of intestinal stenosis, their seat and arrangement furnish valuable and, as a rule, the most important aid in answering the question in which part of the intestine the stenosis is situated. I go still further since, upon the average, the determination of the seat in chronic stenosis of the intestine is easier than giving an opinion in regard to its anatomical nature; as is shown by the above compilation of the anatomico-etiological conditions, certain processes which cause stenosis occur very frequently in individual portions of the intestine, in others very rarely or not at all, and by determining the seat we indirectly gain, at the same time, certain points of support for the recognition of the anatomical process. *For this reason, I generally advise that we first, if possible, determine the seat of the obstruction, and then its nature.*

Descriptions of the various forms of intestinal stiffening and peristalsis which occur in individual cases are hardly possible; illustrations convey a much clearer idea of the condition. I shall, therefore, limit myself to the enunciation of a few general principles.

As a rule, the arrangement and position of the intestinal areas correspond with the normal.

Distention of the flanks and of the upper abdominal regions primarily points to the colon. If the flanks are not at all distended, if they follow a horizontal line, or are even concave, stenosis of the colon may, with great probability, be excluded; but the possibility of congenital or acquired anomaly of position must always be considered. The inverse, however, is not true; i. e., that extension of the flanks may be caused by tensely filled and prolapsing coils of the small intestine. The diagnostic importance of extension of the flanks is especially convincing when the obstruction is situated in that portion of the colon from the hepatic flexure to the splenic flexure; here the right flank alone is distended. In the majority of cases of chronic stenosis, the dilated, stiffened or extremely peristaltic large intestine is in its normal situation (while the enormous degree of inflation in acute obstruction, especially in sigmoid volvulus, creates the most remarkable topographical positions).

Abnormally situated intestinal coils, with alternating and disappearing peristalsis or stiffening, indicate the small intestine. The same is true of several (2, 3 or more) adjacent, oblique or vertical (organ-like) coils upon the longitudinal axis of the body.

The most extreme thickening, as a rule, must be referred to the intestinal portion situated nearest the obstruction, which is accompanied by marked muscular hypertrophy. An exception to this occurs only when the intestine has become paretic by long-continued over-distention.

The fact is noteworthy that movements in the colon are usually slow, in the small intestine they are more rapid and irregular. It is often noted that they cease in a definite locality, with absolute regularity, for example, in the ileo-cecal region, and if there is irregular peristalsis of the small intestine, if the lateral walls invariably remain quiet, we conclude that the obstruction is in the region of the cecum.

These relief-like circumscribed inflations and changes in the shape of the abdominal surface may, however, be entirely absent. The seat of the stenosis is then much more difficult to decide, but it may occasionally be made with certainty, provided physical examination reveals such relations as are calculated to influence directly or indirectly the permeability of the intestine. To this category belong *the proof of tumors* of a neoplasmatic or inflammatory nature, changes in position of the solid abdominal organs, tumors from invagination, chronic peritoneal bands, and old external hernias.

The results of percussion and auscultation are less valuable in deciding upon the seat, are only to be utilized most cautiously, and then rather in cases of occlusion; in stenosis little or no value can be attached to this method of examination.

Vaginal exploration is an important aid, and even of greater significance is *digital rectal examination,* by which we may directly demonstrate the obstruction which produces the stenosis. These examinations should never, under any circumstances, be neglected, for with the regular *examination of the hernial rings* they become a duty upon which too great stress cannot be laid.

Pain is a very unreliable symptom. Only when it is closely circumscribed, accurately located, of uniform degree, and permanent, can we trace its seat to a definite locality, and not always then.

The stenoses of the duodenum and the rectum call for special discussion.

Chronic stenosis of the duodenum gives rise to varying symptoms, according to whether the narrowed area is situated above or below the common gall-duct and the pancreatic duct. The clinical picture in the former case so closely resembles that of stenosis of the pylorus that a differential diagnosis is impossible. Where the constriction is deeply situated there is also gastric dilatation; in this case, however, there are charac-

teristic symptoms in addition. Bile flows profusely and continuously into the stomach so that massive bilious vomiting occurs; or, after gastric lavage, large quantities of pure bile may flow from the tube. Pancreatic juice may also be present in the stomach, so that in this organ duodenal digestion with distinct splitting of fat may take place.

Stenosis of the rectum is easily and most positively diagnosticated, as, in the majority of cases, it may be reached by the palpating finger. Sometimes suspicion is awakened by the apparently causeless development of hemorrhoids and hemorrhages; also by tenesmus. In the main, general diagnostic laws are here applicable.

The employment in our special case of the rules which have been briefly sketched forces us to the view that the seat of the stenosis is in the lowest part of the ileum, in the neighborhood of the ileo-cecal valve (the details of proof may be omitted, and we may assume the diagnosis to be correct since later, when laparotomy was performed, this view was confirmed).

Another question arises: *What is the nature, what the anatomical composition, of the obstruction which causes this stenosis?* The determination of the anatomical nature of the intestinal obstruction is hedged in by enormous difficulty, and, in our opinion, forms one of the most perplexing points in diagnosis. Only the most careful consideration of each individual symptom enables us to proceed, and we should never forget that each concrete case may deviate from the symptomatologic type.

The general rules must be first mentioned.

Primarily, the hernial rings should be examined. Hernias are not only of great importance in acute intestinal occlusion, but old hernias may also form a focus for chronic constriction by means of peritoneal adhesions which lead to constriction and volvulus of the intestine.

Exact digital investigation of the rectum and vagina must be made; this occasionally furnishes the direct clue to the diagnosis, or points the way to it.

If examination of the hernial rings, of the rectum and vagina, is negative, the abdomen must be carefully palpated. In a certain number of cases a tumor will be found, and we must decide whether it be a malignant growth or chronic invagination, a fecal mass or an inflammatory infiltration. The points of support operative here cannot be described. Naturally tumors, particularly those not circumscribed, may be present and escape detection, either because they are too small (which is particularly the case in carcinoma), or because they are concealed by inflated coils of intestine, or for other accidental reason.

Occasionally we succeed in palpating isolated, thicker, peritoneal bands of irregular contour. If we are dealing with tough, massive, distributed and chronic, inflammatory adhesions, such, for example, as are produced by tubercular peritonitis, the anatomical situation clears at once.

If, however, no clue is found which may directly lead to the recognition of the anatomical nature of the obstruction, then an explanation must be sought in the history, in the course of development, and from other circumstances. Chronic inflammatory processes in the pelvis, a preceding perityphlitis, and similar anamnestic data indicate peritoneal fixation, dysenteric and other ulcerative intestinal processes point to cicatricial strictures, very insidious development of the symptoms of stenosis without preceding disease indicates carcinoma. Under these circumstances, it is of special importance in the diagnosis to determine in which portion of the intestine is the narrowed area.

Let us apply this mode of investigation to our special case.

The hernial rings are free. Palpation of the abdomen reveals the intestinal rigidity and increased peristalsis which have been described, which justify the conclusion that the seat of the obstruction is the lowest portion of the ileum—but, beyond this, absolutely nothing to assist in explaining the nature of the obstruction, no palpable tumor or peritoneal band, not even an area painful upon pressure. On the other hand, vaginal and rectal examinations yield positive findings: In the left vaginal vault, a prominent crescentic cicatrix, on the right side a narrow cicatricial band extending to the posterior wall of the vagina. These cicatricial adhesions render a positive palpation of the adnexa difficult. Palpation by rectum shows the pelvis to be filled with markedly inflated intestinal coils, and, besides, particularly upon the right posterior surface of the uterus, posteriorly toward the ligamentum latum, thin, tendinous, coarse bands of adhesions may be distinctly felt. A *preceding pelvic peritonitis* must, therefore, be diagnosticated.

On account of these conditions, the opinion naturally forces itself upon us that these chronic peritoneal processes arising from the internal genitalia are connected with intestinal stenosis, the latter being due to constriction or volvulus. In connection with the otherwise entirely negative findings, this conclusion must be recognized as logical, and no direct, forcible objection to it can be raised.

Nevertheless, we must be careful not to assume without more ado that this conclusion is correct. This caution is not based upon forcible reasons in the particular case, but, on the contrary, upon general experience gained by much observation at the bedside, which may be expressed by saying *that we cannot be too cautious with these diagnoses.* Any one who has observed many of these cases knows how often and how readily mistakes and diagnostic errors arise, even when everything has been carefully investigated in logical sequence. My method of reasoning in such a case is the following:

The diagnosis obtained by direct analysis, it is true, primarily and immediately leads to the presumption, just expressed, of an intestinal constriction or volvulus which is the result of peritoneal adhesions arising from

the genitalia. But—as has been taught us by quite analogous cases—something else might be present, and it is advisable to keep these other possibilities always in mind. What conditions must be considered here? Basis of support is given by two factors: The very insidious onset, and the seat of the stenosis in the lower portion of the small intestine. Both these facts coincide with the assumption of cicatricial stricture or neoplasm. In fact long experience has taught me to formulate the following law: When there is an unquestioned stenosis of the ileum, and absolutely no point by which we may recognize its anatomical nature, we should primarily consider cicatricial stricture following tuberculous ulcers of the ileum, even although no other evidence of tuberculosis can be determined. I have seen conditions exactly as in this case; i. e., stenosis of the small intestine in women in whom peritoneal bands originating from the genitalia were palpable, yet nevertheless, at the operation or autopsy, not these but cicatricial strictures after healed tuberculous ulcers of the ileum were shown to be the cause of the stenosis; or, a small neoplasm, either carcinomatous or tuberculous, not palpable, situated in the lowest portion of the ileum or at the ileo-cecal valve, might also be considered.

From this framework the diagnosis must be evolved; we cannot express ourselves more definitely or more carefully. *Our diagnosis, therefore, in this case is as follows: Peritoneal constriction or volvulus, or a tuberculous cicatricial stricture or neoplasm* (*carcinoma, tuberculoma*).[1]

PROGNOSIS

The prognosis of constriction of the intestine is always serious although not always equally so. The gravity is determined less by the seat than by the nature of the obstruction. In some forms, although naturally very rarely, spontaneous recovery may result; a benign polypoid neoplasm, even an invaginated portion of the intestine, may be thrown off, an exudate which causes pressure may be resolved, a uterus in abnormal position may be replaced, and thus the cause of the stenosis is removed. But these are all exceptions.

Without treatment stenosis almost always causes serious consequences. If no complications occur, the course of a benign stenosis is as follows:

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[1] I here include the following:

Laparotomy revealed the existence of pelvic peritoneal bands; but these were chiefly found in the large intestine, and there was no causal connection with the stenosis of the ileum. In the ileum not one but two stenoses were found, removed about 6 cm. from each other. A portion of the intestine about 13 cm. in length was excised; examination revealed two ring-shaped, coarse, fibrinous, cicatricial strictures. The mesentery belonging to this coil of ileum contained glands which were hard and the size of a hazelnut; microscopic examination revealed tubercular disease. Accordingly the intestinal strictures, also, must be looked upon as originating from tuberculous ulcers.

For a long time the compensatory hypertrophy of the musculature overcomes resistance, making the propulsion of the intestinal contents possible. Gradually, however, insufficiency of the musculature appears; it becomes paretic; fecal stasis follows, and finally, all the consequences which characterize intestinal occlusion—operation or death. In other cases the changes in the mucous membrane above the stenosis determine the further course. Diffuse peritonitis or sacculated fecal abscesses may result from decubital ulcers, also severe septic conditions, or perforation with its fatal issue. In carcinomatous stricture the process is hastened by the malignant nature of the growth.

Here and there intermediate conditions may add to the constriction and lead to sudden and complete occlusion. Thus, solid foreign bodies (fruit-stones, asparagus) or even a coarse particle of feces may completely occlude the narrow opening of the intestinal lumen. Or the heavy intestinal coil above the stenosis which is filled with feces may descend, causing torsion of the axis and volvulus. If the antecedents are unknown, or have been disregarded, cases of this kind may occasionally simulate acute intestinal occlusion. Sometimes the true condition may be suspected if active peristalsis and rigid stiffening arise, for these permit the conclusion of hypertrophy of the intestinal musculature; therefore, of an obstruction which has existed for a long time.

TREATMENT

In conclusion we must discuss the treatment.

This may be instituted with two objects: To limit the disease to a *symptomatic course*, or to attempt the *radical removal of the obstruction.* We shall begin with the latter.

Except in very isolated cases the removal of the stenotic obstruction is possible only by surgical measures. Fecal masses in the interior of the intestine narrow it but do not cause total occlusion; an inflammatory exudate capable of resorption gives rise to external pressure; these belong to the greatest rarities. In such instances treatment must be instituted in accordance with the well-known laws which apply in the concrete case. In others only the knife can aid us; it is superfluous to explain this in detail, for a glance at the previously described anatomical causes of constriction proves this convincingly. The question is only this: When shall we operate? The answer to this must be: As soon as the diagnosis of constriction has been made with certainty. For, if left to itself, the condition can never ameliorate but steadily becomes more serious. In the case of carcinoma, operative interference as early as possible is, in fact, our duty, for, apart from the consequences due to the stenosis, the danger of metastasis from the prolonged existence of the neoplasm constantly increases, and the chances of a successful operation constantly decrease. In eica-

tricial strictures and in constricting indurations we are not influenced by this latter consideration, but even a speedy operation is indicated, as a spontaneous recovery is not to be expected under any circumstances. Nevertheless, here we may more reasonably temporize a little, and only insist upon the operation when the clinical conditions of the pathologic picture (unbearable pain, transitory intestinal occlusion) make it necessary.

Symptomatic treatment must be based upon three different points of view: The period of time between the individual attacks of colic, the paroxysms themselves, and the appearance of complete intestinal occlusion.

The first object is attained by regulating the diet and evacuating the bowels. Large quantities of food should never be taken at one time. Its quantity must be definitely arranged; everything is to be absolutely avoided that is unsuitable in volume to the digestive powers, or which might even itself produce an obstruction, such as rye-bread, slices of potato, fibrous and tendinous meat, above all, most vegetables and fruit with their skins and seeds. It is best only to give pappy, finely divided, or finely chopped food; in the more marked grades of stenosis only fluid or semi-solid food.

It is clear that a regular daily evacuation of the bowels must be carefully brought about. According to the seat of the stenosis serviceable remedies are irrigations (of water, oil, or soap and water) or purgatives administered internally, among which the neutral salts and purgative waters, preparations of senna or rhamnus and colocynth are preferable.

The attacks of colic necessitate the complete withdrawal of food and the employment of purgatives; if active peristaltic movements and intestinal stiffening which show activity above the stenosis are present, irrigations but no cocoprotica by the mouth are indicated. Where the pain is unbearable, warm compresses to the abdomen, and occasionally even an opiate must be resorted to.

A further indication may be the appearance of complete occlusion. It is of the greatest importance that we understand clearly in the individual case how this serious result has arisen, as very different measures may be necessary in the relief of this condition.

If the stenosed area is still permeable, and only accidentally occluded by compact intestinal contents, irrigations and salines which reduce the intestinal contents to a fluid may relieve this condition.

If paresis of the musculature above the constriction is the cause of the so-called symptoms of ileus, to these evacuants we add the employment of electricity and massage, also gastric lavage, and a trial of the atropin treatment which will be described more in detail in the article upon complete intestinal occlusion. These non-surgical measures must be carried out with a clear conception of the anatomical conditions and functional disturbances. Their employment is limited to a very brief period. As soon as stasis of the intestinal contents assumes a threatening aspect, particularly as soon as the cardiac action begins to weaken, operative interference affords

the only hope of saving life. This should never be long postponed, even although strangulation of the intestine have not supervened, which, in acute occlusion, causes very fulminant symptoms; a possible cardiac insufficiency should always be taken into consideration. What the operative process would be in a concrete case cannot be here described.

II. INTESTINAL OCCLUSION

The second history which I shall relate refers to an acute invagination of a portion of the intestine into an adjacent part. This process led to the complete closure of the intestinal lumen, and produced a symptom-complex which is usually grouped under the designation "ILEUS." As this symptom-complex depends not only upon mechanical closure of the intestine but also, in spite of complete permeability, upon paralysis of the musculature of the intestine, in the former case we speak of "mechanical," in the latter of "paralytic" or "dynamic" ileus. The clinico-symptomatologic limits of this term are by no means exact or sharply defined. It appears to me that occasionally there is danger in the use of this elastic term, inasmuch as by its use in a concrete case the impression may be given that the diagnosis has been clearly established. For this reason, I think it wise to reject the term ileus, although it is undoubtedly useful on account of its brevity. In its place, however, in every case where possible, not only a functional but an anatomical diagnosis should be made.

PATHOLOGY

The pathology of intestinal occlusion is much more complicated than that of intestinal stenosis. In a single concrete case it is impossible to analyze all of its features; therefore, in describing this condition I shall pursue a method different from that used in presenting stenosis.

Let us then begin upon the firm foundation of the *anatomical changes.*

In occlusion the permeability of the intestine is completely arrested. Neither compact, nor fluid, nor gaseous contents pass downward. As in stenosis a marked contrast exists between the intestinal coils situated above and those below the occlusion; the picture, however, is not uniform, but varies according to the causal relations of the process.

In those cases in which a slowly progressive stenosis gradually leads to complete occlusion, the same anatomical condition is found which I sketched in describing stenosis; often there is decided thickening of the intestinal wall, with catarrhal and ulcerative implication of the mucous membrane above the area of occlusion.

On the other hand, in acute occlusion which occurs suddenly in an intestine previously healthy, the distention of the upper intestinal coils is occasionally enormous, but the wall is not thickened by muscular hypertrophy; on the contrary, according to the nature of the anatomical process

causing the condition, it is sometimes thin and pale, at other times dark blood-red and succulently thick. The distention is usually caused by a so-called *stasis meteorism*, i. e., by stasis of the intestinal contents and gas above the occluded area. The intensity and distribution of the same toward the stomach varies in individual cases; if the seat of the occlusion be in the colon, the resistance of the ileo-cecal valve is overcome without difficulty.

Wahl and Zoege v. Manteuffel have recognized, in contrast to the usual form, another variety, the so-called *local meteorism*. This appears when, besides the narrowing of the intestinal lumen, severe circulatory disturbance in the sense of a venous stasis simultaneously occurs in the intestinal wall and in the mesentery; this is most frequently observed in torsion and kinking, occasionally also in internal forms of incarceration resembling hernia. Stasis meteorism may happen in these forms, but may also be absent. In the latter case, the intestines above and below the area of occlusion are absolutely empty; on the other hand, the coils between the points of occlusion are dilated (particularly in volvulus and in kinking), they are dark blood-red, their walls are thickened by an accumulation of fluid; gas, blood and fluid fill the interior—and this condition in particular is designated as local meteorism. In complicated noose formation empty and local meteoric coils may vary, and present anomalous pictures. The development of gas is due to the *strangulation*, i. e., to the venous circulatory disturbance which, in the previously mentioned varieties of occlusion, is found in the intestinal wall and in the mesentery (an implication of the mesenteric nerves in the strangulation may also be possible). In spite of the fact that some authors still raise objections to the assumption of a local meteorism, it nevertheless appears to me that this certainly occurs if the conditions necessary for its development are present; and its exceptional absence in spite of the latter may be explained by special circumstances in the individual case.

Other anatomical changes, in so far as they present themselves in the various forms of peritonitis, will be considered under the description of the clinical picture.

To obtain a clear conception of the clinical picture of complete intestinal occlusion, a few fundamental laws must be first laid down.

While stenosis always comes on slowly (with the single exception of an acutely arising invagination which merely causes stenosis of the lumen but not entire occlusion) and insidiously, occlusion in quite a number of cases appears suddenly in the midst of perfect health, and only in those cases in which it is due to stenosis do the symptoms gradually appear.

Even more significant is the following law:

In some cases the symptoms are the result of phenomena produced by the entire cessation of the passage of feces (*symptoms purely of occlusion*). This usually occurs when the occlusion develops from stenosis.

In other cases *symptoms appear which are to be attributed to strangulation*, and these consist of disturbances of the circulation in the intestinal wall and in the mesentery combined with toxic symptoms and those dependent upon derangement of the nervous system. The clear recognition of this condition is not only necessary for the understanding of the pathological process, but in a high degree will also decide the therapeutic indications and methods.

I shall first describe the symptom-complex of pure occlusion, i. e., the *symptoms which characterize simple occlusion*; this, as a rule, comes on slowly and gradually and is caused by a cicatricial or carcinomatous stricture, or, more rarely, appears acutely, in consequence of the impaction of foreign bodies or gall-stones.

SYMPTOMS

The passage of retained gas as well as of compact and fluid masses ceases entirely. Absolute constipation follows and the complete cessation of flatus per anum. Particles of feces which may perhaps be expelled by irrigation proceed from that part of the intestine below the occlusion and have been retained in parts of the colon.

The patient is attacked by severe pain. This presents itself as a painful attack of colic such as I have described in the article upon stenosis (which see), and in which energetic peristalsis and, occasionally, extreme intestinal rigidity become visible and palpable. In the acute cases of intestinal occlusion—and this is a valuable diagnostic point—these conspicuous symptoms are absent, and the conditions for their development, particularly decided muscular hypertrophy, do not exist. Therefore, in most of the cases in which the intestine shows excessive and marked muscular hypertrophy with symptoms of occlusion, we may suspect an anatomical process that has gradually produced occlusion from stenosis.

Step by step, deficient appetite, nausea, eructation and vomiting appear. Usually the gastric contents are evacuated, and then bile. The vomited matter soon reveals a feculent odor, and finally the dreaded *fecal vomiting* takes place.

Fecal vomiting—it may be remarked in passing that some authors employ the term "ileus" only when this symptom appears—on account of its offensiveness has been duly studied from the earliest times. The feculent character of the vomited material is readily diagnosticated by the sense of smell. The color is usually a dirty yellow, or it may be greenish or brownish. Almost invariably it is fluid, and resembles watery diarrheic discharges. That more compactly formed feces have been discharged by the mouth, as has been positively reported by some observers, cannot be denied, but such cases certainly belong to the greatest rarities, and then should be analyzed individually, as these disagreeable symptoms may be simulated by malingerers and hysterical individuals. Sausage-like masses

resembling formed feces may be discharged from the mouth, but more minute investigation reveals that they are composed of coagulated milk the surface of which has been stained yellow by bile. True, formed feces may be vomited in consequence of the rare occurrence of a communication between the stomach and the colon; but this phenomenon cannot be considered as a parallel with fecal vomiting in intestinal occlusion.

The opinion previously maintained that fecal vomiting denotes positively that the seat of the occlusion is in the colon, or, at least, in the lower portion of the ileum, has for some time been recognized as erroneous. We now know that this symptom may arise even in occlusion of the upper part of the jejunum. The explanation is simple. The disagreeable odor caused by the decomposition of albumin bodies, and which lends to the intestinal contents their feculent character, occurs also under the influence of definite varieties of bacteria. A prolonged action is necessary for this, such as occurs normally in the large intestine, but not, however, in the contents which rapidly pass through the small intestine. If, on account of occlusion, the contents are retained for a long time in the small intestine, here also they may denote a feculent composition. *Vice versa*, this explains how fecal vomiting may be absent even with absolute occlusion of the intestinal passage: namely, when death occurs very early, or when severe vomiting does not permit prolonged stagnation.

As to the manner in which fecal vomiting occurs, there is unanimity of opinion. In the first place, it is not caused by a *motus antiperistallicus*. It is true there is actual antiperistalsis, but the requirements for its development under the conditions here operative are not present. A detailed exposition of this interesting question is at this point impossible. Usually the explanation given by Haguenot-van Swieten is accepted. If the intestine is occluded, more or less rapidly the portion above the occluded area is filled with gas and fluids furnished by the gastric contents and the secretions which enter the intestine and attempt to pass onward. When the stasis reaches a point immediately above the obstruction, formed by the distended intestinal wall, the retained mass is gradually forced upward in the direction of the stomach. Vomiting now occurs which may be due to any cause. During this act the diaphragm and abdominal muscles contract, the area of the abdominal cavity diminishes, and pressure is exerted upon the intestine and its contents. The volume of the fluid contents cannot be propelled downward toward the anus on account of the insurmountable obstruction, nor can it be pressed upward, and thus, in a retrogressive manner, it is forced toward the stomach, and fecal vomiting results. It is readily perceived that, analogous to the act of vomiting, the contractions of the intestine, moving in a normal direction, must mechanically affect the contents in its lumen, thus causing a regurgitation of the contents. That the act of vomiting as such, and not antiperistalsis, is the cause of the expulsion of the intestinal contents is borne out by an expe-

rience of my own that in an enterostenosis of the sigmoid flexure the entire colon and the small intestine up to the jejunum were filled with thin feculent contents, yet fecal vomiting did not occur. The patient did not vomit during the entire course of the disease and retching even was not present.

Meteorism occurs, but by no means in the same form and to the same extent in all cases. This depends upon the etiologico-anatomical form of the intestinal occlusion. It is true that the passage of flatus ceases entirely, but the more important resorption of gas from the intestine into the blood may continue as long as a simple stenosis of its lumen without an alteration of its wall exists, and in case the formation of gas due to the accidental presence of abnormal quantities of fermentative material does not preponderate over the absorption of gas. Under these circumstances meteorism will be but moderate. This is more frequently observed in occlusion which develops from stenosis of the intestine; quite as frequently also in the case of internal occlusion of the intestine (*obturatio intestinalis*) by foreign bodies, gall-stones, intestinal stones and fecal masses; and also in cases of external compression. On the other hand, in the forms of acute occlusion which run their course with strangulation of the intestinal wall and of the mesentery, marked degrees of meteorism may be observed. Here the areas nearest the occlusion show the greatest distention, and from the peculiar formation and situation of the inflated coils important data not only for the localization of the seat of the obstruction but occasionally even indicative of its anatomical nature may be obtained.

Finally, as a direct symptom of occlusion, the *increased excretion in the urine of indican and other combined ethereal sulphates* must be mentioned, of which indicanuria is the most important in practice because of the readiness with which it may be chemically determined. In occlusion of the small intestine there is a large, and occasionally enormous, increase of indican in the urine; in occlusion of the colon only when this lasts a long time, and when there is stagnation of its contents above the ileo-cecal valve. In the use of this symptom embarrassment has arisen from the fact that under some conditions which must be here considered, very frequently, in addition to the occlusion and also as produced by it, a diffuse peritonitis which runs its course with decided indicanuria is superadded.

All of the symptoms which have as yet been described are the direct or indirect consequence of the arrested impermeability of the intestinal lumen, and they appear when the cause of the impermeability in its nature has this effect only, and does not, at the same time, cause strangulation. Only in isolated cases, especially in acute obstruction by large gall-stones, do other symptoms appear which resemble the severe clinical picture of strangulation.

We shall now turn to these.

It must first be remarked that the *clinical picture in which symptoms of occlusion and strangulation appear simultaneously* is, without excep-

tion, produced by acute processes. These are acute strangulation, volvulus, noose formation, and particularly also invagination. Each of these forms presents typical peculiarities which are not characteristic in every concrete case. But the main features of the clinical picture are common to all, and these I shall now enumerate.

Suddenly, sometimes in the midst of perfect health, and, in any event, without preceding abdominal disturbance or external cause, occasionally after a prior mild or indistinct intestinal disturbance and pain, or following jumping, a fall, or a blow, the patient is seized with severe pain in the abdomen. This is sometimes localized, at other times diffuse; occasionally persistent, at other times remittent and colicky. To this is added nausea, vomiting, singultus. The vomiting may then sooner or later assume a feculent character. Sometimes (in invagination, in volvulus) tenesmus appears. If, after the pains set in, a movement of the bowels occurs, the passage of feces and gases subsequently ceases entirely. Meteorism appears, sometimes moderate, at other times intense, according to the nature and the seat of the occlusion; there may be extreme distention of definite configuration.

The *symptoms of strangulation* are added to these symptoms of occlusion which, except for their usually rapid development, externally resemble those before described, although pathogenetically they differ somewhat.

A serious condition now sets in, which profoundly implicates the whole constitution. The expression of the face denotes anxiety and pain; the nose, the ears and extremities are cool and livid; the pulse is frequent, small, and easily compressible; the respiration is often rapid and superficial. There is a feeling of oppression; the secretion of urine ceases. Yet the mind retains its clearness. Unless nature or art comes to our aid the disease progresses to its highest degree, the skin becomes withered and flaccid, is cool and livid and often covered with cold sweat. The features are sunken, the pulse thready and extreme prostration is the prelude to death.

This is an outline of the clinical picture of acute strangulation, but the individual symptoms must be briefly analyzed.

A merely superficial glance permits the recognition of two groups of symptoms: On the one hand the gastrointestinal, and, on the other hand, phenomena dependent upon the nervous system and upon the circulation.

Of the former group of symptoms, the majority are unquestionably the direct and mechanical consequences of occlusion; namely, the absolute stoppage of the bowels, the passage of flatus, and the occurrence of fecal vomiting. On the other hand, the initial simple vomiting, the pain, and the meteorism may have an indirect cause, and not be exclusively and immediately the consequences of occlusion of the intestinal lumen.

The *initial pain* is one of the most invariable and conspicuous symp-

toms of acute occlusion, and is rarely absent in incarceration, in volvulus, and in invagination. If localized and fixed, it owes its origin to a sudden, local irritation of the nerves of the intestinal wall; this is also true of the colicky pain which occurs in paroxysms, of the energetic peristalsis which increases and decreases, and of the tetanic intestinal stiffening. Besides the pain which is localized, there may be more diffuse pain which is also initial, which is not increased by external pressure, and which is not as yet the consequence of peritonitis. This is caused by the irradiating stimulation of the large ganglion plexuses and, according to my experience, is more severe in occlusion of the small intestine than in that of the large intestine, more severe in incarceration, which usually attacks the ileum, than in volvulus which is most common in the sigmoid flexure. In complete acute intestinal occlusion even without peritonitis, the pain is almost always constant, although there are remissions and exacerbations; as the lethal termination approaches, it sometimes ceases in consequence of intestinal paralysis.

In the onward course of the disease, *vomiting*, although not yet feculent, must be considered so pathogenetically. The *initial vomiting* which occurs at the onset of acute occlusion, and the hiccough which appears simultaneously, are unquestionably of reflex origin, and due to the sudden, severe irritation of the nerves of the peritoneum and of the intestine. This process of development explains the constancy of vomiting in acute occlusion of the small intestine which is richly supplied with nerves, and in this form of the disease it is rarely absent.

I have previously mentioned that in the development of meteorism in occlusion combined with strangulation, not only in stenosis of the intestine a cause but also the cessation of gas resorption from the lumen of the intestine in consequence of alteration in its walls. This alteration in the intestinal wall occurs in the area of the strangulation, and accounts for the fact that in this form of occlusion a peculiar configuration is occasionally met with which has been designated above as "local" meteorism.

This is most characteristic in acute volvulus, which occurs with greatest frequency in the sigmoid flexure, and to such an extent that volvulus, either in its own longitudinal axis or in the axis of the mesentery, is greater than in any other portion of the intestine. In volvulus of the sigmoid flexure, as a rule, local meteoric distention in the sigmoid flexure sets in very rapidly, and after forty-eight to seventy-two hours it may attain such enormous dimensions that the entire abdomen appears inflated like a balloon. This inflation begins in the first hours of the disease in the left lower abdominal region; the inflated coil then extends toward the right and upper abdominal areas, and finally distends the entire abdomen to a globular form. Nowhere is peristalsis observed; the percussion note over the distended region is tympanitic, exceptionally dull, the latter condition being explained by the circumstance that the walls of the intestine are

markedly edematous and thickened, and the contents of the sigmoid flexure consist of blood and feces. In the other forms of acute occlusion which accompany strangulation, namely, internal incarceration and invagination, local meteorism either does not occur at all or scarcely develops to such an extent as to be of clinico-diagnostic importance.

Although it has been already mentioned, the importance of the subject leads me to reiterate that in acute occlusion, if this attack an intestine previously permeable, peristalsis that is distinctly visible and tetanic intestinal stiffening only occur exceptionally, and never to the extent seen in occlusion which follows a slowly growing stenosis. The reasons for this are obvious: In the first place, there is no muscular hypertrophy of the intestinal wall in acute occlusion, and the paralysis of the musculature which occurs develops with particular rapidity in the doubly occluded and strangulated intestinal areas in which local meteorism has developed; paralyzed muscles, of course, do not contract.

The extremely rare occurrence of *diarrhea* in acute occlusion (in invagination, incarceration), is deserving of brief mention, since, at first, it may lead to gross errors in diagnosis. This is explained by the hypothetic assumption of a hypersecretion of fluid from the intestinal wall in consequence of paralysis of the mesenteric nerves.

Besides this group of symptoms, other symptoms on the part of the circulatory apparatus are noted; these dominate the situation, and impress their stamp upon the affection, the clinical condition closely resembling "collapse" from other causes. The extent of these phenomena may vary in individual cases; as a rule, however, they are decidedly marked.

In regard to their occurrence two views are maintained at the present time: A nervous reflex theory, and an intoxication theory. In my opinion the latter is operative only for certain rare symptoms and cases. On the other hand, the nervous reflex theory readily explains the majority of symptoms which are clinically common and important, and which usually appear at the onset of the affection.

I cannot here analyze the reflex theory in detail, but will only remark that in accordance with well-known physiological facts the severe clinical picture is caused by decided irritation of the sensory nerves of the intestine and of the peritoneum; that is, of the mesentery, which results with the acute onset of volvulus, incarceration, invagination, obstruction from gall-stones, etc. Occlusion thereby leads to an alteration of cardiac activity and of the entire distribution of the blood, this being brought about by the reflex influence of the cardiac vagus and of the splanchnic nerve. In fact, the majority of the symptoms may be thus explained, particularly those above described and also some rare ones; as, for example, the albuminuria and cylindruria which are occasionally observed in incarcerated hernia.

The idea of the "intoxication theory" is this, that in the closed in-

testinal passage the arrested products of bacterial proteid decomposition which had formed in large amounts (phenol, indol, skatol, the aromatic oxyacids) are absorbed by the blood, and thus exert their toxic action. A modification of the intoxication theory, designated as "septic collapse," assumes that the intestinal microbes reach the peritoneum through the intestinal wall, which is damaged by the strangulation, and thence, even without causing peritonitis, infect the entire organism. The following may be stated in criticism of these views: It is possible that a few very rare symptoms of strangulation shock may be referred to intoxication of this kind, and among these delirium or coma, rise of temperature without peritonitis, and the so-called "incarceration typhoid." But the facts have by no means been determined with certainty, and against its general acceptance very important clinical objections may be raised; for instance, that the severest picture, the most profound collapse, may develop in from six to twenty-four hours after the occlusion; that, as a rule, the intoxication is in proportion to the suddenness of the incarceration and the severity of the initial pain; that in occlusion which develops gradually it may be entirely absent.

The course of the disease is not always the same; it varies according to the anatomical foundation, the cause of the occlusion, and particularly according to whether simple occlusion or accompanying strangulation exists.

In occlusion with strangulation occurring suddenly, death may follow in a few hours (eight) with all the symptoms of collapse. At other times peritonitis is added and brings about the lethal termination. Its development in these forms is favored by the fact that in the area of the strangulated region there is a histological alteration in the intestinal wall which makes it possible for microbes to penetrate into the peritoneal cavity. To this already alarming picture more circumscribed or distributed spontaneous painfulness upon pressure is added, according to the distribution of the peritonitis. This peritonitis may, although rarely, develop in pure occlusion. On the other hand, if no relief is brought about, death usually occurs by gradual exhaustion in consequence of the absence of nourishment and of continuous vomiting.

What are the conditions which lead to complete intestinal occlusion?

I have already mentioned quite a number of the causes under intestinal stenosis (which see). All, or nearly all, of the processes there enumerated are capable, either directly or indirectly, of causing complete occlusion of the intestinal lumen. They furnish the cases in which occlusion forms, as a rule, by gradual stages from the constantly advancing stenosis, and in which the clinical picture is composed of the symptoms of pure occlusion. Exceptionally, and under special circumstances, the stenosis suddenly and rapidly increases to entire impermeability; as when a coarse for-

eign body entirely occludes the narrow passage, or the dilated portion of the intestine above the stenosis, which is filled with contents, descends and leads to volvulus and the like.

A *second series* of cases is furnished by complete occlusion of the intestinal lumen by any large impacted masses there present (obstruction of the intestine). Of this variety are:

(a) Enormous fecal masses which, in addition to their direct constipating effect, produce a paralytic weakness of the musculature of the intestine;

(b) Intestinal calculi;

(c) Gall-stones;

(d) Foreign bodies.

In this series of cases the clinical picture exhibits exclusively the symptoms of occlusion. Only in obstruction due to gall-stones are symptoms of collapse occasionally observed which, as stated previously, are produced by the severe sensory irritation of the stone upon the intestinal wall.

A *third series* of cases is formed by processes which not only cause occlusion but, at the same time, produce strangulation of the intestine, that is, of the mesentery:

1. Volvulus;

2. Noose formation—which, at this time, is usually included under the designation volvulus;

3. Hernia, internal incarceration;

4. Invagination, intussusception.

These conditions almost invariably occur acutely, and are accompanied by the distressing symptoms of the clinical picture last mentioned.

In conclusion, an affection must be mentioned which mechanically has not the remotest connection with occlusion of the intestinal lumen, but whose physiologic termination is nevertheless the same.

This is *paralysis of the intestine*. Here, the lumen is wide open, the passage mechanically unhindered; nevertheless, its function has ceased because motor paralysis in a large or small portion of the intestine makes the propulsion of the contents through this area impossible.

We require no explicit description to make it clear that, besides these conditions which in combination form the clinical picture of occlusion, or of occlusion with strangulation added, each individual one may produce in the clinical picture its own distinctive symptoms. In this article, which is merely a comprehensive review of intestinal occlusion, it is impossible to enter more minutely into the subject. Each of the various conditions would require special description.

DIAGNOSIS

Although the analysis of the symptoms which make up the clinical picture of occlusion is simple, in reality it may be extraordinarily difficult to make the *diagnosis at the bedside.* In fact, when the individual points are considered, and occasionally even in deciding the fundamental question, this proves a most difficult task for the physician. The most experienced and the most careful investigator is liable to err, and yet, in this grave affection, not only is certainty but, especially, rapidity of diagnosis of the utmost importance in the treatment.

In each individual case the diagnosis must be based upon the answers to three distinct questions:

1. Is intestinal occlusion present?
2. In what portion of the intestine does it exist?
3. What is the anatomical foundation of the condition?

In regard to the first question: *Is intestinal occlusion present?* Errors in diagnosis may be made in two ways. Intestinal occlusion is assumed because the symptom-complex is more or less completely developed; yet, notwithstanding, this condition is not present, but the symptoms are due to another affection. Or the symptoms, which but partially indicate the condition, divert the attention of the physician into another channel, and the actually existing occlusion is overlooked.

The diagnosis is based mainly upon the local intestinal symptoms: Absolute impermeability for gas as well as feces, meteorism, vomiting, fecal vomiting.

That the first three symptoms, taken individually, furnish but limited proof need not be dwelt upon. Even fecal vomiting is of no pathognomonic diagnostic value. Apart from its possible appearance in hysteria, and from its actual but very rare occurrence in fistulas between the colon and stomach, it may also appear—as well as absolute constipation, meteorism, and vomiting—in intestinal occlusion from paralysis of the intestine and from peritonitis (in the latter instance also it is pathogenetically due to intestinal paralysis). Under the following circumstances, the physician must consider the possibility of pure intestinal paralysis without mechanical occlusion: When fecal vomiting appears after some dull trauma has affected the abdomen; when it persists in incarcerated hernia after the mechanical obstruction has been removed by the knife or other means; in general, when it appears after surgical operation upon the intestine itself or in its immediate vicinity; or when it has developed in disease of the testicles, in the inguinal region, in the inflammation of hemorrhoids, in renal colic, in gall-stone colic, in recent acute appendicitis and perityphlitis. To this must be added the possibility of the appearance of intestinal paralysis in embolism of the mesenteric artery, in habitual constipation, in over-distention from excessive meteorism and, perhaps, also in

consequence of bacterial peritoneal infection without peritonitis. Under all these circumstances fecal vomiting and the entire symptom-complex *may* be caused by pure intestinal paralysis, and the physician must always bear this possibility in mind, and should never forget that in the concrete case actual mechanical occlusion may also be present.

The most frequent and the greatest perplexity is caused by the question: Occlusion or peritonitis, or their combination? Its vital importance constrains me to dwell somewhat more explicitly upon the individual points which may aid us in the differential diagnosis.

The pain is of great significance. Severe pain occurs spontaneously in acute peritonitis as well as in acute occlusion. If to this, however, diffused, well-developed tenderness of the entire abdomen upon palpation is soon added, if the patient lies very quiet and carefully avoids voluntary movement, this is more in favor of peritonitis. Naturally, the latter condition may very rapidly develop in occlusion, but scarcely sooner than after forty-eight, or, at the earliest, twenty-four hours. The possibility of examination within the first or the second day may, therefore, give us important data to clear the diagnosis. Of course absence of pain upon pressure is by no means absolute proof of occlusion and disproof of peritonitis, for, on the one hand, even in acute and purulent peritonitis spontaneous pain as well as pain upon pressure may be only of minimal degree, and, on the other hand, the rapidly developing meteorism in volvulus and in internal incarceration may occasionally run its course with quite marked sensitiveness to pressure; nevertheless, decided painfulness upon pressure more strongly indicates peritonitis.

Sometimes in acute cases the degree of meteorism will establish the diagnosis. If, at the onset, it develops decidedly as local meteorism, if it is possible to detect circumscribed, inflated, extremely tense portions of the intestine by simultaneous percussion and auscultation, we may assume volvulus or an internal incarceration. Diffuse meteorism, on the contrary, may be observed in occlusion as well as in peritonitis, and for this reason cannot be utilized in a differentio-diagnostic respect: the enormous balloon-like inflation makes an accurate examination impossible.

The significance of the intestinal movements in such conditions has been several times described. I believe it to be certain that positive, decided peristalsis and, particularly, paroxysmal intestinal stiffening always shows an obstruction in the passage. Inversely, if it has been determined after careful examination by means of inspection, palpation, and auscultation that there is absolutely no sign of intestinal movement, this points with great likelihood to peritonitis with intestinal paralysis. But the occurrence of intestinal movement is, unfortunately, not absolute and positive proof. For complete stoppage may also occur without peritonitis in consequence of enormous over-distention, and, inversely, it may continue even after the development of peritonitis.

The presence of a fluid exudate is in favor of peritonitis, but here it is necessary for us to be cautious; since, on the one hand, in internal incarceration, particularly in volvulus in consequence of mesenteric venous stasis, such a profuse hemorrhagic transudation may occur in the peritoneal cavity that a peritoneal effusion is simulated; on the other hand, the latter may be actually present, but, because masked by peculiar physical conditions, may not be recognized.

Fever points to peritonitis; but the absence of the same does not exclude peritonitis, as any form of this affection may sometimes run its course without fever and even with subnormal temperatures.

Occasionally the presence of indicanuria may be of diagnostic importance, naturally only under very definite conditions. The positive proof of increased indicanuria coincides with the existence of a diffused peritonitis as well as an occlusion of the small intestine. However, its absence is against the presence of either of these processes, and the same is true of an occlusion of the large intestine, for example, volvulus of the sigmoid flexure.

Naturally other factors may be utilized in the diagnostic consideration of the concrete case. For example, the presence of florid ulcerative processes in the stomach or intestine is an indication of peritonitis. Careful analysis will very frequently reveal the true situation, but there are cases in which the diagnosis, either of peritonitis or intestinal occlusion, may be impossible.

If the presence of intestinal occlusion has been determined the question next arises: *In what portion of the intestine does it exist?* To answer this is extremely difficult. As a positive aid it is well first to determine clearly whether the occlusion has developed from stenosis or whether its onset was acute, that is, simultaneously with symptoms of strangulation. In the former case its localization is readily decided, and as guides, the factors which I have mentioned in the article upon intestinal stenosis, to which I here refer, may be utilized.

If we are dealing with acute occlusion, that is, with strangulation, the most minute manual exploration of the hernial rings, as well as of the rectum and vagina, is necessary. Occasionally, by this means, important data are revealed, particularly in incarcerated external hernias.

Irrigation of the rectum, which is much practised in order to determine the seat of the occlusion from the amount of fluid which may be injected, is sometimes impracticable on account of the condition of the patient, and even when possible the result is frequently unreliable. A deep-seated obstruction may be determined only when, after repeated irrigations, but little fluid can be forced into the intestine, and this invariably returns rapidly. But we must not forget that the rectum with its ampulla may take up from one to one and a half liters of fluid. Sounding of the rectum is also unreliable.

In acute strangulated occlusions, local meteorism in the individual case may occasionally enable us to come to a decision in regard to the seat of the occlusion, particularly if volvulus of the sigmoid flexure is present and the development of meteorism is observed by the physician himself, who can refer it to the coils of the sigmoid flexure. The points of support which we assume from the functional symptoms on the part of the intestine are, as a rule, susceptible of various explanations and untrustworthy.

In insidious occlusion there is sometimes entire absence of pain. In the acute forms, naturally, it is usually present, but only when it is narrowly circumscribed, definitely fixed, persistent, uniform, and localized, should pain be utilized as a guide, and even then cautiously; and I regard its importance in localization as of greater significance when it is not only spontaneously present but may also be developed in the same area upon pressure. The law that in acute occlusion of the small intestine the pain sets in earlier, more severely, and is more continuous than in that of the large intestine, is in itself subject to many exceptions, and to this may be added that in volvulus of the sigmoid flexure also the pain may be exceedingly severe.

Vomiting may be of importance in the diagnosis in so far that generally—concrete exceptions may occur in either direction—in acute occlusions of the small intestine it is more regular, more continuous and severe than in those of the large intestine. Fecal vomiting, as I have previously remarked, may occur in any localization, even in high-seated jejunal occlusion.

Very little, but occasionally something, may be learned from the state of the feces. Marked tenesmus may occur when the seat of an acute occlusion is in the lower colon. An admixture of blood is more apt to be observed in this condition when the occlusion extends from the cecum far downwards.

The value of indicanuria as pointing to the seat of the affection may be formulated in the following manner: If, in a previously healthy person, the symptoms of acute occlusion (but without symptoms of peritonitis) appear, and if upon the second or third day marked indicanuria is present, this points to the small intestine and is against the large intestine. With a prolonged duration of the occlusion, its absence also favors the large intestine, its presence, however, no longer furnishes any clue.

Some general view-points have been made use of in deciding upon the seat of the occlusion. It was formerly believed that with a very rapid course and markedly developed symptoms of occlusion the seat might be referred to the small intestine. This is, however, absolutely incorrect, for, if the seat be in the small intestine, the symptoms at the onset need not necessarily be very severe, and *vice versa*. In volvulus of the sigmoid flex-

ure the symptoms may rapidly appear and be exceedingly severe. If, in the concrete case, the anatomical nature of the occlusion is readily perceived, this may sometimes also facilitate the recognition of its seat.

The third part of the diagnosis, namely, *the determination of the anatomical nature of the pathological process*, is sometimes easy, usually difficult, and, in a few instances, impossible. Nevertheless, we should always attempt to decide it, and for this purpose the following method has been found useful:

The hernial rings, which should be first examined, and the rectum sometimes directly furnish the anatomical diagnosis (hernia, intestinal invagination).

If an old hernia exist, even although it be not directly (incarceration, inflammation) and solely the cause of the symptoms, nevertheless its utilization must be held in reserve until investigation discloses another undoubted cause (volvulus or internal incarceration from peritoneal adhesions originating in the hernia).

If hernia or an obstruction in the rectum be excluded, the history must be accurately reviewed. If this denotes chronic stenosis from which complete occlusion has finally developed, the same examination and considerations must be pursued as in the diagnosis of that form of the disease which I have already described in stenosis of the intestine.

If we are, however, dealing with acute occlusion in an individual previously healthy—acute peritonitis, naturally, does not here come into question—the following anatomical conditions are possible: Obstruction by foreign bodies or from gall-stones, volvulus, internal incarceration, invagination, intestinal paralysis.

The view of obstruction from foreign bodies must be at once rejected or maintained according to the history (except in insane patients). In obstruction due to gall-stone, the history almost invariably furnishes data which point to gall-stone disease; moreover, we have usually the picture of occlusion and not of strangulation. If, as often occurs, the history furnishes no points, and if, besides, the symptoms of collapse be added to those of occlusion, for instance, such as are produced by the sensory irritation of the stone upon the nerves of the intestine, a diagnosis becomes impossible.

Volvulus, internal incarceration, invagination, all, as a rule, set in with severe pain and vomiting. The same is true of acute peritonitis. The perplexity, which sometimes cannot be overcome, in differentiating from acute peritonitis, which begins in the same manner, I have already emphasized and have indicated the steps toward a possible solution. The factors which may lead to the assumption of intestinal paralysis—under the picture of occlusion—have also been previously mentioned. These two possibilities, acute peritonitis and intestinal paralysis, must always be minutely considered. If there is no foundation for their diagnosis, if the

conditions incline us to the assumption of volvulus, of incarceration, or of invagination, other points of support must be present to make possible the differentiation of these processes.

Pain is here of the less importance, because it is common, almost invariable, and usually the initial symptom in these conditions. Perhaps its primary seat may indicate the anatomical diagnosis: If extending from the left hypogastrium to the sigmoid flexure, volvulus; if in the right hypogastrium, to an invagination of the ileum and cecum; if in the middle and upper abdominal region, to an internal incarceration or volvulus of the small intestine.

Even less significant is vomiting.

Absolute constipation is the rule in internal incarceration, in volvulus, and in obstruction from gall-stones, sometimes too in invagination, but here diarrhea occasionally alternates with constipation. Hemorrhagic discharges are most frequently observed in invagination, rarely in volvulus of the sigmoid flexure, very exceptionally in internal incarceration, and not at all in obstruction due to gall-stones. The same laws exist for tenesmus as in regard to hemorrhagic discharges.

Collapse suddenly appearing may occur in all forms of occlusion, most markedly in internal incarceration, next in volvulus, and to a less degree (even entirely absent) in invagination and obstruction.

Better grounds for the diagnosis may be furnished by the local findings in the abdomen, if it be possible to obtain these. Above all, acute local meteorism may lead to definite conclusions. If we can be sure that the tensely inflated, elastic, motionless, intestinal coil rises from the left lower side of the abdomen, this is in favor of volvulus of the sigmoid flexure. Invagination may be determined with certainty if a tumor is present which possesses the characteristic properties of an invagination tumor; namely, that it is usually soft but periodically becomes hardened by rigid contractions of the wall.

TREATMENT

Among the most difficult and responsible duties of the physician is the treatment of intestinal occlusion. Upon his decided opinion, based directly upon his definite conclusions, naturally, the life of the patient often depends. While this opinion in the case of occlusion developing from chronic stenosis occasions no immediate anxiety, the situation in acute occlusion often necessitates most urgent measures; within a few hours the most far-reaching decisions must sometimes be made, and this oftentimes when we only indistinctly recognize the seat and the nature of the occlusion, and occasionally are even in doubt as to whether an occlusion or, on the contrary, a paralysis of the intestine is present. Consider the distressing picture with its characteristic features—features mostly uniform and which repeat themselves—when the patient, collapsed

and vomiting, is found to have complete intestinal obstruction, his abdomen distended like a drum, and all diagnostic points which aid in the determination of the nature of the affection are absent! Nevertheless, the physician must act. The patient, unless already in the terminal agony, must not be left to his fate. What is to be done?

Some authors absolutely deny the occurrence of a spontaneous cure, that is, without surgical interference, in an internal incarceration, a volvulus, or kinking. Others, upon a basis of actual experience, believe this to be possible in favorable anatomical conditions and in the earlier periods of the affection, although exceedingly rare. The first contingency, however, absolutely requires operative interference; the latter permits, within definite limits and even here in a certain period of time, non-surgical treatment. Naturally, the latter does not and cannot decide the treatment, and this statement is only made to show how a very surprising course may be occasionally explained.

In regard to treatment, we must differentiate between simple occlusion and occlusion with strangulation.

In occlusion with strangulation it is the rule to operate as soon as the symptoms of the latter become at all marked. If the diagnosis of the seat and the nature of the disease can be made, exploratory laparotomy and removal of the obstruction is in place. The chances of operation are best if it be done within the first forty-eight hours after the appearance of the symptoms of occlusion: 75 per cent. recover; after the third day, the percentage of recoveries falls rapidly to 35 to 45 per cent. It remains on this plane even when the operation is done only in the course of the second week.

If the physician recognizes that the intestinal occlusion is either internal incarceration or volvulus, no time is to be lost by attempting any other method of treatment. For the psychical effect upon very anxious patients and to relieve the minds of friends and relatives during the time when preparations for the operation are being made, at most only a brief trial of non-surgical measures is allowable.

It appears that twists, if not of marked extent, may occasionally recover under non-surgical treatment. This may also be resorted to in invagination, but in these two conditions operation should be attempted early, provided non-surgical measures do not rapidly bring relief.

If gall-stones cause intestinal occlusion, experience has shown that the outlook for a spontaneous recovery or by internal remedies is not unfavorable. In this case, we should decide upon laparotomy only when the symptoms indicate direct danger to life. The gauge for this is furnished by the cardiac activity; we shall revert to this later.

What has been said of gall-stones is also true of large and round foreign bodies. When sharp or angular substances cause obstruction purgatives must be avoided, and in their place soft food should be taken and potatoes,

rice, oatmeal grits, which form a mass safely enclosing the foreign bodies.

I shall now return to the question of operation.

Experience teaches that the *chances of operation* not only depend upon its early performance, but also increase with the recognition of the nature and location of the obstruction, as this makes possible a direct attack at its seat. If, however, the diagnosis is obscure, and only peritonitis and intestinal paralysis are excluded, what is to be done? The answer must certainly be: Operate at once, provided the operation in itself would be a harmless affair. This is, however, not the case if symptoms of strangulation are at all marked. Although the modern surgeon may, with some degree of certainty, exclude septic infection in peritonitis, other dangers threaten, particularly in these laparotomies, and especially if the seat of the disease must first be searched for (intestinal paralysis, reflex influences upon the nervous system and upon the circulatory apparatus). Here, that is, with an uncertain diagnosis, two kinds of cases must be differentiated. If the course is very acute, if there are severe symptoms of shock, probably only the operation can save life. Without this almost all patients perish, and one saved by this means may be looked upon as pure gain. If, however, the course is more moderate, the entire picture not too severe, so-called internal treatment may be resorted to with a definite view and purpose which shall be later explained.

In attempting this therapy, the physician must carefully observe the *cardiac activity,* as shown by the quality of *the pulse.* In these cases the condition of the pulse will correctly indicate the best time in which to perform the operation. If the pulse begins to increase in frequency, the tension to sink but a little, it is time to operate, for somewhat later this may become impossible. The weakened cardiac activity is no longer sufficient to meet the requirements of an operation. In such cases it is much better to operate several days before this critical cardiac change than to wait but a few hours after it has taken place.

If the symptoms of occlusion exclusively appear in the clinical picture, while those of strangulation are absent, often an operation alone can save the life, but in the less fulminant course of the affection there is time to try non-surgical measures. In quite a number of cases these may entirely relieve the threatening situation.

There has been much discussion of late as to the cases of intestinal occlusion in which a resort to non-operative treatment is at all permissible, under what circumstances, and as to the factors upon which the decision depends.

Primarily, a point of support, as I have already mentioned, is the general condition as expressed in the cardiac activity and in the condition of the pulse, while the local symptoms of occlusion (meteorism, vomiting) are of less import. But, I repeat, if the diagnosis at the onset is un-

certain, and if the cardiac condition is good, relief by non-surgical measures may be attempted, for, during this time, by rapid decision, we may obtain more data in support of the local and collective diagnosis to improve the chances of operation. If by the application of these principles the possibility and justification of a brief delay is gained, the question then arises which non-surgical measures are most likely to be successful.

First, a method which is *not* to be resorted to must be pointed out. In all forms of acute, subacute, chronic, and complete intestinal occlusion *purgatives are decidedly dangerous*, and for this reason must be absolutely avoided—with the single exception, in case the obstruction is due to fecal masses. With a positive or lightly acute occlusion by incarceration, torsion, kinking, or obstruction by gall-stones and foreign bodies, as well as whenever the diagnosis is questionable, purgatives should never be employed! Only when in a concrete case obstruction due to feces is almost certain may purgatives be administered by the mouth. Mercury, formerly so much used, may be entirely dispensed with, for it is occasionally directly harmful and still more so indirectly, as valuable time is lost in its employment.

Enemata and rectal irrigations are often advised, but very little more may be expected from them than from purgatives by the mouth. If they do not flow off again they also have a harmful effect, since by their massiveness they raise the already increased intra-abdominal pressure. These facts must always be taken into consideration. *A priori*, and according to experience, enemata and large injections are useless in nearly all forms of acute occlusion; the only exception to this is formed by acute invagination which, if it have occurred in the large intestine, may, in fact, be caused to disappear by forced massive enemata or by the introduction of air. In one form of chronic occlusion these are almost indispensable, and combined with internal purgatives they form a most valuable therapeutic agent, namely, in obstruction from feces, immaterial whether this takes place in a free atonic intestine or above the point of stenosis. But we must be very careful in their use if there is a well-founded suspicion of ulcerative processes or fissures in the intestine.

For irrigation, water (1–5 liters) or oil ($\frac{1}{2}$–1 liter) is employed. The temperature of the water should be that of the air; ice-water injections are no more beneficial, and are contraindicated in all debilitated patients. Highly irritating additions, particularly of table salt (5 to 8 per cent.), are perhaps only of use in acute invagination. The value of the irrigation depends, in the main, upon its softening property, and for this purpose pure water, or soapy water, or oil is sufficient. The fluid must always be introduced with only slight force and very slowly, from one-half to one hour being required.

Lately carbonic acid enemata have been employed, of which about the

same is to be said as of injections of water. The inflation of gas into the rectum, as occasionally practised, is naturally useless in obstruction due to feces; in the acute forms of occlusion the same is true as of the irrigations with water, i. e., an effect is only occasionally to be expected in acute invagination.

Massage of the abdomen, as well as the application of the faradic current in the milder degrees of the affection, may prove useful in a single form, namely, that of obstruction due to feces. On the contrary, massage as well as faradism is to be absolutely rejected in all forms of acute occlusion with strangulation, and also when the possibility of peritonitis or hernia may be assumed, which, in the case of strangulation, often begins very early. In invaginations of very recent origin, massage (with simultaneous narcosis) is said to be occasionally useful; but even here the greatest caution is necessary and, in my opinion, rectal injections and inflations are generally more successful and considerably less dangerous.

An important enrichment of our therapy in acute intestinal occlusion, *gastric lavage*, has been introduced by Kussmaul. Its value consists in this, that the evacuation of the gastric contents facilitates the regurgitation of the intestinal contents into the stomach. This causes the cessation of retching and fecal vomiting and a subjective feeling of amelioration is produced. Experience shows, however, that the employment of gastric lavage occasionally does even more than this; an absolute cure of the severest forms of complete intestinal occlusion in cases lasting from eight to nine days has been brought about by its use. This treatment should not be deferred until too late, and we should never rest content with a single lavage, but the method must be employed frequently, often being repeated every two or three hours, since the regurgitation of the contents of the small intestine into the stomach may take place with extraordinary rapidity. The same objection has been made to gastric lavage as to opium treatment, that it brings about a deceptive euphoria while the local process remains unchanged or advances. As stated, these agents occasionally have a curative effect, and, apart from this fact, the patient who suffers severely must not have such a beneficial remedy withdrawn from him even although it have but a symptomatic effect. For it is the duty of the intelligent and conscientious physician constantly to keep in mind the question of an operation, and, in spite of apparent subjective improvement, closely to observe the most decisive local symptoms (passage of feces and flatus) as well as to watch over the cardiac activity.

I must still refer to two drugs used internally. One of these is *opium and its preparations*, which may be administered either by the mouth or by subcutaneous injection in the form of morphin. There is great difference of opinion as to their use. That they may be given, or must be given, to tide the suffering patient over a few hours of misery when the situation is hopeless and operative interference is no longer possible, is

generally recognized. In some cases its employment is permitted by even its most bitter opponents; for instance, if in high-graded stenosis, enormous peristalsis and intestinal stiffening make us fear early paralysis of the musculature of the intestine, and if, during this time, attempts have been made to overcome the obstruction by rectal irrigation and other measures. The dispute, on the contrary, is concerning the question whether or not opium may be given, that is, must be given at the beginning of acute occlusion. Some surgeons are decidedly opposed to its use for the following reasons: Because the treatment by opium brings about only an apparent improvement, and by masking important symptoms renders the diagnosis difficult, increases the danger of collapse, and facilitates the onset of intestinal paralysis; also because the patient, on account of apparent improvement, rejects an operation. In opposition to this the following may be said: The feeling of intense anxiety, the nausea and vomiting, the severe pains, frequently cease. If given at the right time, i. e., immediately at the onset, opium occasionally has a surprising effect upon the symptoms of initial reflex collapse. And even if this were its only effect—which is not the case, as it occasionally influences peristalsis, even, perhaps, makes possible the spontaneous improvement of the anatomical disturbance—treatment by opium should not be rejected for it brings great relief to the suffering patient. The dreaded deleterious effects may be obviated if, as I have just remarked concerning gastric lavage, the physician does not permit himself to be deceived, and continually keeps in mind the local intestinal symptoms and the cardiac condition; if, moreover, the employment of opium is instituted only upon the first or second day of the disease, and, notwithstanding its apparently good effect, the question of operation is decided according to the principles which I have indicated.

The other remedy, quite recently introduced into the treatment of "ileus," is *atropin* given subcutaneously in decided doses, 0.001 to 0.005 at a dose, and repeated if necessary. In the last few years a series of cases has been reported in which atropin was used under most desperate circumstances when for many reasons an operation was out of the question; it freed the intestinal tract, and recovery followed. It is impossible to base an opinion upon the statistics at hand. In the cases in which atropin was ineffectual, an anatomical intestinal occlusion was shown. In the single case in which I have had opportunity to observe its favorable effect, the symptoms were those of severe intestinal occlusion in consequence of obstruction due to gall-stone. I presume that the effect of atropin in intestinal occlusion may best be observed when the underlying condition is that of a spastic or paralytic obstruction.

In acute occlusion the *ingestion of food* must be absolutely suspended from the first day. When the thirst is tormenting small pellets of ice may be given by the mouth or minimal amounts of ice water may be swal-

lowed. In case of profuse loss of fluid by vomiting, fluid may be injected into the rectum or subcutaneous infusions of normal salt solution may be employed. Where the course of the disease is prolonged, nutritive enemata are to be advised.

For severe cases of well-developed intestinal paralysis the measures previously described (massage, electricity) have proven ineffectual. Further experience will show whether physostigmin (physostigmin salicylate, 0.0005 per dose), recently introduced by v. Noorden, will furnish any better results.

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